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THE POINTING POSITION OF THE HAND AS A PATHOLOGICAL AND PRIMITIVE REFLEX.[1]

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OUR symbolic expressive movements may be divided into two distinct groups. To one of them belong gestures which clearly show their origin in primitive biological behaviour. Thus the movement of threatening with the clenched fist is nothing but the beginning of an attack. If the affect increases, the threatening with the fist may change into a blow against the adversary.

In contrast with these gestures, representing the beginning or the decrease of a purposive movement, are others, no less frequent, the relation of which to some biological situation is not clear. Why, for example, has the military salute the well-known form of elevating the extended right hand to the forehead? Have we to do here with mere convention founded on tradition, or is this gesture based upon biological or physiological mechanisms which are preferred to other motor complexes in the given psychical situation? Though we may believe those gestures which we use as a kind of colloquial language to have at least a local social historical cause, yet in most cases we are unable to trace the biological origin of such symbols.

In the following pages I will try, starting from some ontogenetic and pathological observations and experiments, to explain how it is that when indicating some object we do so with the characteristic position of pointing: right arm fully extended, the back of the stretched hand upwards with the index extended and the other fingers flexed towards

[1] By the kindness of Prof. Kurt Goldstein, I have had an opportunity since this manuscript was finished of seeing the proof-sheets of a paper of his pupil, Hans Cohn (Weitere Beobachtungen über Haltungs und Bewegungsstörungen bei Schädigung des Stammganglienapparates." "Zeigestellung," Mitbewegungen, "induzierte Tonuserscheinungen," already published in *Zeitschritt für die gesamte Neurologie und Psychiatrie*, Bd. 125, H. 2/3, S. 327.) This paper also mentions the provocation of the "pointing position," but does not discuss the reflex character of the phenomenon.

the palm, either clenched to a fist, or hanging inactive. I was led to this problem by the observation that the characteristic position of pointing, which we make as a voluntary movement, which is excited automatically as a synergy, or which is seen in various extrapyramidal lesions as a spasm (see below), may also be induced by a definite method as a reflex.

This observation was first established in the following case :—

F. H., a boy aged 10. He was said to have "trembled" from birth, and to have sudden transient accesses of pallor at the age of 8. He appeared to be imbecile, and had never walked or talked normally. There was temporal pallor of the optic discs. The pupils were unequal and immobile. Corneal and conjunctival reflexes were absent. Visual acuity seemed to be normal. Articulation was defective and the protruded tongue was restless. There were jerking movements of the head and limbs, variable muscle tone and increased tendon-jerks. Co-ordination was defective with intention tremor and ataxy. The right plantar response was extensor, the left was not obtained. Sensation appeared normal.

The pointing position of the hand was best elicited with the patient propped up in bed, the arm flexed and pronated and supported on a pillow, and the fingers almost fully extended. The patient was told to shut his eyes, and the endeavour was made to divert his attention from the procedure to be described. The index finger was then stretched by the observer. Thereupon the remaining digits actively flexed, the middle finger least, the little finger most. On releasing the stretched index, the pointing position of the hand was maintained for a brief interval. Other passive movements of the index evoked a similar response: as rotation of the index on its long axis. Moderate degrees of passive lateral deviation of the index (ulnarwards or radialwards) evoked a less active response than stretching (fig. 1).

The converse reaction, namely, active extension of the index when the remaining digits are passively flexed on the palm, did not occur. Pin-prick, pressure, thermal and electrical stimuli applied to the extended index did not elicit the "pointing position" response, i.e., active flexion of the remaining digits.

Thus the characteristic pointing position of the hand was elicitable only by deep sensory stimuli arising in the neighbourhood of the joints of the index finger, comparable stimuli to the other digits being without effect.

The phenomena thus described were observed in the right hand, but a less ample and slower response of the same nature was also obtained from the left hand. In both cases undue frequency of stimulation led to fatigue of the response, more readily in the left than in the right hand.

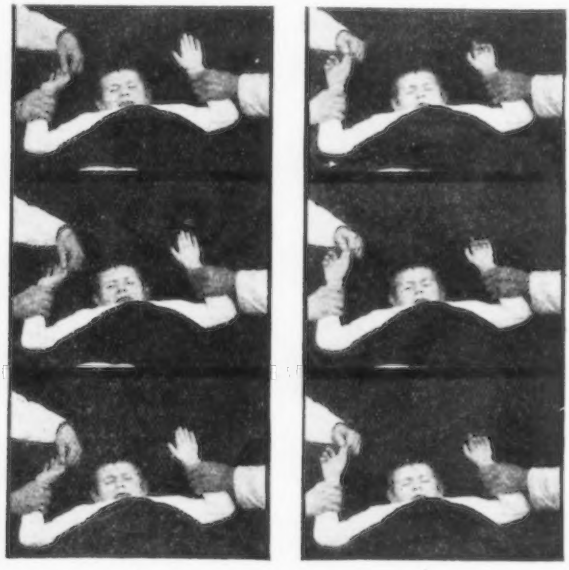
Under favourable conditions, as after rest, it was often observed that if the pointing position were provoked on the right side, the same position simultaneously, though less obviously, appeared in the contralateral hand (fig. 2). Scarcely any movement of the index in its distal

FIG. 1.—Passive extension of the index induces a "pointing position" of the hand; the three ulnar fingers and the thumb assume flexed positions while the index remains in active extension.

joint was to be seen, but a distinct flexion of the other fingers could be observed, the thumb going into a flexion-adduction position. I did not succeed in exciting the crossed phenomenon on the right side from the

left hand. The contralateral "pointing position" fatigues even more rapidly than the ipsilateral phenomenon, and after three or four repetitions the mechanism is exhausted, recovering relatively early from fatigue. When the patient's arm was held passively in a flexed position for a longer time, slow movements of the fingers set in, which resembled the athetotic movements described by O. Förster [3] : "slow alternating movements of flexion and extension of the fingers" [1].

These movements might perhaps be interpreted as voluntary movements of the fingers resulting from an uncomfortable position of the



a *b*

FIG. 2.—The beginning (*a*) and the end (*b*) of a series of photographs which show the appearance of the "pointing position" in the opposite hand.

arm. The regularity of their rhythm, the stereotyped positions assumed, and their regular production by passive movement of the index indicate involuntary activities.

We have first to decide whether we are dealing with a reflex or not. A similar question has been raised by Carl Mayer [12], especially in relation to the finger, "Grundgelenk-Phänomen," that has since become an accepted aid in the diagnosis of lesions of the pyramidal system above the level of the cervical cord [5].

We need not consider, in discussing the "pointing position," the objection that the "Grundgelenk-Phänomen" is not a reflex, but a conscious pain reaction (A. Meyer), for the adequate stimulus of the "pointing position" does not excite pain, while painful stimuli do not elicit it. Nor can it be due to a mechanical effect without participation of the centrally excited muscular contractions, for in normal adults even a violent passive stretching of the index does not excite bending of the other fingers. Moreover, the fact that we can provoke the pointing position contralaterally shows it does not depend merely on mechanical relations.

That it is not a purely voluntary movement is proved by the fact that in our case the phenomenon was always more distinct on one side of the body than on the other, and that after a short series of succeeding reactions fatigue ensues, disappearing quickly after resting he hand.

Nor is the pointing position a result of a withdrawal movement or something of that order, for painful and thermal stimuli provoking withdrawal do not excite it. Further, stimuli which excite the pointing position at the index remain without any effect when applied to the other fingers.

The selective stimulus necessary to evoke the pointing position as well as the contralateral response, the exclusion of a voluntary element and of mechanical factors, all indicate the reflex character of the pointing position. We may therefore call it the "pointing reflex." It is a co-ordinated multi-muscular reflex, in which by a specific stimulus coming from a certain region (index) of a functionally connected group of muscles (fingers of the hand) simultaneous movements are excited. It leads to a position (pointing position) which can also be assumed as a voluntary purposive reaction.

In order to determine its reaction time, the reflex was cinematographically recorded, the film being moved with constant velocity (sixteen pictures per second). The stimulus was the application of a forceps to the index; this was easily visible on the film, and the number of the pictures between the closing of the forceps and the first visible change of position of the ulnar fingers relative to a fixed point therefore gives approximatively the latent time of the reflex.

This interval averaged between three to five pictures, so the latent time is about 0·25 second. As the latent time of joint reflexes is 0·20 to 0·25 second, and of tendon reflexes, as the patellar, about 0·05 second [1], the reaction time of the pointing reflex belongs to the first class.

This points to also the reflex nature of the reaction, and suggests that it may be a reflex which can be influenced by cerebral mechanisms.

We must take into consideration here the co-existence of an "Eigen-reflex," as described by P. Hoffmann [7]. That the reaction is not a simple "Eigen-reflex," is shown by the fact that the latter is unilateral only, shows practically no fatigue, has a short reflex time and appears only as part of a "fremdreflectorisch" or voluntary movement. The pointing reflex has functionally a position intermediate between voluntary movements and the relatively fixed extrapyramidal motor disturbances, which also occasionally give rise to the typical pointing position.

It may be emphasized that, according to O. Förster [3], athetotic movements of the hand in the striate syndrome show a tendency towards flexion, ulnar adduction and pronation. The similarity of these movements to the position of the hand in pointing towards a distant object is obvious.

K. Goldstein and W. Bornstein [6] describe a case, probably with striatal disturbances, in which the right hand on becoming rigid assumed the pointing position, while after recovery from the spasm involuntary movements appeared, which produced the same characteristic position of the fingers. In another patient, aged 14, who suffered with epilepsy after injury of the head, the authors state that the pointing position occurred as a final tonic attitude during the epileptic fit [13].

A. Meyer [13] likewise observed the typical pointing position in a child with cerebral infantile paralysis of the right arm during the tonic phase of the epileptic fit. Similar cases reported by Pötzl, Schloffer and Lowenstein are cited by Meyer.

In a picture given by A. Jakob [9] we can see this pointing position of the hand in a somewhat exaggerated form as a contracture in a case of paralysis agitans.

I have observed this pointing position as a habit position ("Habitual-Stellung"), in several normal children between 2 and 4 years of age, especially when the arm had been stretched against gravity.

In the following case I was able to show, under certain conditions, an interesting transition between the pointing position as a reflex phenomenon and as a spontaneous habit position.

The patient was an epileptic, aged 20. His physique was infantile and poorly developed. His intelligence was that of a boy of 8 years, and there were epileptic changes in his character. Since childhood he had frequent fits

without any aura, in which he fell to the left, his eyes turned to the right and his limbs were convulsed. His pupillary reflexes and vestibular reactions were normal. There were variations of muscle tonus in his arms and legs, athetotic-ataxic movements of his outstretched fingers, and Meyer's thumb phenomenon was more pronounced on the right than left. Ankle clonus could sometimes be obtained on both sides, but the plantar reflexes were indefinite.

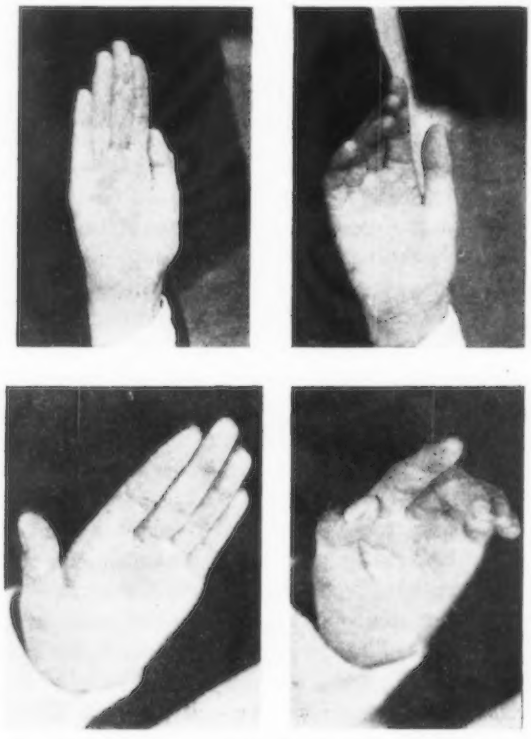


FIG. 3.—The "pointing position" appears in the one hand when the fingers of the other hand perform a series of active movements.

The pointing reflex could be obtained only when the tonus of the muscles was normal and the fingers relaxed. If the arm were moved about passively the muscles contracted tonically and the hand extended involuntarily; the pointing reflexes could not be then obtained. I found, however, that this tonic contraction could be prevented by active and passive movements of the opposite arm, and I could then obtain the pointing reflex.

The patient was seated and both arms were supported on a soft cushion. A screen was placed in front of one hand so that he could not see it. When

made to open and close the hand he could see involuntary movements of the other hand occur, first flexion and extension of the little finger, then of the fourth finger, and finally all fingers flex at the metacarpophalangeal joints so as to approximate to the position of a closed fist.

When he tapped on a board a rhythm chosen by himself the fingers of the other hand assumed slowly the pointing position, the little finger flexed first, and then the fourth and third in succession, and the thumb became opposed. This occurred most readily when the arm was supinated.

If he voluntarily assumed the pointing position with either hand the fingers of the other hand passed into a similar position. This occurred whether the arm was pronated or supinated, but in the latter position the fingers flexed at their basal joints only, and the whole hand became more rigid than when the arm was pronated (fig. 3).

These observations show that voluntary movements of one hand in this patient excited movements and attitudes of the other hand which are similar to the pointing position. The movements excited in the resting hand tend to correspond to those in the moving limb. When the reaction is feeble the attitude tends to the pointing position, when strong to the grasping position.

This influencing of the tonus of one extremity by contralateral activity accords with observations I have described in which a provoked increase of the tonus of one arm causes a shifting of the tonus equilibrium from one side to the other, and disturbances of the innervation of the contralateral arm.

The pointing position can obviously be excited by motor mechanisms of different levels, and it therefore probably represents a functional unit. Goldstein and Bornstein [6] have come to the same conclusion from their investigations of analogous spasms, but the question whether there is a special "localization" of the pointing mechanism cannot be discussed here.

Further observation with children of different ages who already have a pointing synergy and are able to point to an object on demand, give the following results: If you ask them, e.g., "Show me the glass," when a glass is before them within reach, the first reaction is almost always a grasping and offering of the object. If we forbid the grasping of the object and tell the child to point only, we often see that he involuntarily begins a grasping movement. If the object lies beyond reach the child tends not merely to point to it, but to run to and grasp it and to bring the glass to the experimenter. Only when the child sees that it cannot reach the object it reacts to the question: "Where is that object?" by pointing. We can, however, provoke grasping instead of pointing under the following conditions: the experimenter

arranges a number of objects before the child, so that it can grasp them, and gives his commands as: "Show me the glass," "Show me the apple," quickly in succession. If by this means we make the child perform a series of movements quickly, sometimes involuntary grasping instead of the movement of pointing appears. The pointing position of the hand appears where the act of grasping, which, it may be suggested, is biologically more primitive, is either suppressed or prevented by external conditions. I assume, therefore, that the pointing position is modified grasping, that it probably originated phylogenetically as an enfeebled and modified act of grasping in trying to reach an object which is beyond reach by extension of the fingers and arm. If you tell the child to grasp an object, as a lamp, which is hanging too high, he stretches his hand and all his fingers towards the object. Thus, the pointing position of the hand is an expression of the tendency to grasp under conditions where grasping is impossible. Pointing is partly an enfeebled grasping, partly a means of extending the grasping hand towards the unattainable object.

Considered as a symbolical expressive gesture, pointing is a sublimation of grasping. Phenomenologically, pointing towards an object has also some analogy with grasping it; by pointing towards the object it becomes virtually held.

The evolutional primacy of grasping over the act of pointing may be seen on comparing the muscular contractions and the relations of tonus in the grasping and the pointing positions of the hand. The tonus of the musculature and the degree of flexion of the fingers is greater in grasping than in pointing. The increase of flexion and of tonus during pointing occurs only when pointing energetically towards an object or towards a person. In affective states, as while disputing with somebody, we often point towards our opponent with the index extended and the thumb and the last three fingers closed to a fist. But if the dispute becomes more vigorous the index may flex and the hand assume a position as if grasping at the opponent.

It is interesting that in opening the fist gradually the tonus of the index first relaxes and it extends before the ulnar fingers.

The second, third and fourth fingers form together a closer synergic unit than does any of them with the index. The transition from grasping to pointing must, therefore, begin with the index. It is in advance of the other fingers in the change of position of the hand; while the other fingers are still tonically flexed it has begun to extend. Later, when the other fingers are still slightly flexed, the index is fully and tonically extended.

I regard these relations between the mechanisms of grasping and pointing as an example of a fundamental, functional correlation between all the characteristic movements and positions of the fingers. It may be looked upon biologically as a synergy, occurring very early in evolution and representing a purposive action, like grasping, climbing [2], the movements of rowing [4], &c.

It will be, however, more advantageous to study the development of these motor mechanisms in childhood than to indulge in biological speculations. By observations and experiments on newly-born and older children I found that the pointing position as a habit position and as a reflex movement develops from the original mechanism of grasping. It is well known that the hand of the new-born child generally assumes the fist position, with the thumb covered by the strongly flexed fingers. When the open hand is touched it closes reflexly immediately, the so-called grasping reflex: this is met also as forced grasping and groping in disease of the frontal lobes of the brain and in lesions of the basal ganglia [12], [15], [10], [8].

In newly-born children, who clearly showed the grasping reflex, I could not elicit a pointing reflex. The reason for this does not lie in any mechanical factor, as Trömner [16] states, but in the fact that the grasping synergy of the hand as a whole is still too strong.

The hand in new-born children only moves as a non-differentiated whole. If when the flexion tonus of the hand passes off for a moment, a pull is applied to the extended index, or to any other finger, all fingers flex again into a fist.

The pointing reflex is absent in the new-born child because the grasping reflex is still predominant, and because the tendency to stretching of separate fingers is not yet developed; this occurs only with the anatomical and functional development of the pyramidal system [2].

In normal children of about the age of 2 years, I could always demonstrate a pointing reflex. Up to the 5th or 6th year of age it is a normal phenomenon. The younger the children in whom the pointing reflex was elicited, the more frequently was it observed that reflex flexion of the fingers was excited not only by passive overstretching of the index but also by extension of the other fingers. The pointing position, as a reflex which can be produced only from the index, seems to develop from the grasping reflex. While the tendency to flexion predominates, it may still be excited by non-differentiated stimuli; then voluntary impulses begin to determine the pointing position as a

whole. Later, when the pointing position appears as a habit position, only differentiated stimuli, as applications to the index, provoke the reflex flexion of the other fingers. Finally, these stimuli also lose their excitatory effect, and the physiological total process corresponding to the pointing position is seen only in the characteristic pointing position of the hand which it automatically assumes on voluntary initiation of the symbolical-expressive gesture of pointing.

It is noteworthy that the pointing reflex may be observed at the age at which also the "hand-arm phenomenon" of Leri and the finger-thumb reflex of Mayer appear, that is, according to Goldstein, only after the end of the second year [5]. The end of the second year is also the age at which the normal dorsiflexion reflex of the great toe changes into the normal plantar flexion reflex [11]. It is not yet clear how these coincidences are to be explained, but everything points towards the conclusion that particular mechanisms such as "grasping" and "pointing" form a unity, considered as physiological mechanisms and as modes of behaviour.

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CHRONIC PROGRESSIVE POLYNEURITIS, WITH REPORT OF A FATAL CASE.

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AND

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CASES of chronic polyneuritis may rarely progress so steadily and gradually as to give a clinical picture which superficially resembles that of progressive muscular atrophy. Remissions are the rule, but in the case here reported there was no period of improvement and death occurred about a year after the onset.

Sorgo [8] described a case of recurring polyneuritis in which the first attack of paralysis developed progressively during a period of five months.

Barnes [1] described cases as "toxic degeneration of the lower neurones simulating peripheral neuritis." His Case 1 is of special interest: a female, aged 21, in whom general loss of muscular power followed rheumatic fever and became progressively more extreme during a period of seven months. Bedsores and incontinence developed, with difficulty in swallowing and speaking; improvement then occurred.

Grinker [3] described a case of progressive polyneuritis in a painter, aged 65, who had suffered with colic. Progressive atrophy and paresis of the muscles caused death without remission in nine months. Autopsy revealed changes only in the peripheral nerves. There was no degeneration in the white columns of the cord. There was no shrinkage of the anterior horn cells and no change in the Nissl bodies.

Harris [4] considered that "slow chronic polyneuritis" was the most suitable term for describing this type of case. He reported two severe cases.

(1) A male, aged 31, whose symptoms came on following the appearance of large purpuric blotches on the legs. Some pain in the calves preceded a gradually progressive paralysis which increased in severity for twelve months. The arms were not affected until nine months after the onset. On examination seventeen months after the

onset, there was almost complete flaccid palsy of feet and legs, hands and wrists. All deep reflexes were lost and there was some anæsthesia in the periphery of all four limbs. No cause was found.

(2) The second case he reported was of a girl, aged 15, who became gradually weaker during twelve months until all four limbs were totally paralysed: she was unable to raise her head from the pillow and her facial and masticatory muscles were weak. Apart from slight paræsthesiæ at the commencement, there were no sensory symptoms. No improvement occurred until eighteen months after the onset, when she slowly began to regain power and recovered completely in another two years. In this case also no cause was found.

The case which is the subject of the present study was admitted to the National Hospital for Nervous Diseases on October 10, 1929, under Dr. James Collier.

Mrs. C., aged 45. She complained of paralysis of the arms and legs, breathlessness and spasms of choking. She was hardly able to speak owing to respiratory embarrassment, so the history was mainly obtained from her husband.

Previously she had enjoyed particularly good health. No other illness was known. She had been married for seventeen years. There had been two miscarriages but no children.

Her father died of "old age" at 76. Her mother died in a mental hospital aged 55. Eight siblings were alive and well: none have died.

As to habits, she only rarely takes alcohol, has always a good appetite and takes a varied diet. No contact with any possible poison was discovered.

History of illness.—A year ago she began to complain that everything was too much for her and that she readily became exhausted. About the same time she had a rash over the left side of her face and neck. This began on her neck and spread gradually in the course of six weeks over the side of her face. It then gradually receded, disappearing first from the forehead and last from the neck. There was no temperature or marked malaise; no crusts or oozing. The eye was not inflamed and no scar was left. There was severe local irritation which kept her awake at night.

Ten months ago she would complain that her arms felt weak when washing. Thereafter the muscular weakness became progressively worse. Her knees "gave way" five months ago and since then she has been unable to walk. Her arms also became worse and she was admitted to Redhill Hospital (Edgware) on June 12, 1929 (four months ago), where she remained till August 6, 1929.

NOTES FROM REDHILL HOSPITAL RECORDS.

There was no temperature. The fundi were normal. The pupils were equal and reacted normally. Slight nystagmus was present on looking to the

right. Marked hippus was present. The patient was unable to stand owing to great weakness of the legs. The upper extremities were almost powerless. No contraction of the biceps or pectoralis major could be demonstrated on either side. She was able to move the fingers a little in both hands. Some wasting of the interossei was present.

The faradic responses were normal in the muscles of the arms and legs. All deep reflexes were absent. There was no loss of sensation. The lower limbs were hypersensitive to pressure.

The fractional test meal and blood-picture were normal. No stippling of red-cells was seen.

The Wassermann reaction was negative in the blood and cerebro-spinal fluid. The latter contained one cell per c.mm.

She was seen by Dr. Russell Brain on July 23, 1929, who diagnosed her condition as a toxic polyneuritis.

While in Redhill Hospital she continued to become weaker and her husband took her home, where the downward progress continued steadily. There was no apparent period of improvement throughout her illness. She became completely powerless in her limbs and during the last two months of her life had numerous choking attacks which became more and more frequent. She developed great respiratory distress during these and became very blue. She also had difficulty in swallowing.

Patient stated that when the weakness of the hands commenced it was associated with feelings of "pins and needles," and numbness in both hands.

Clinical examination on admission to the National Hospital, Queen Square.—The patient sits propped up in bed. Her breathing is laboured and her face cyanosed. The respiratory rate is 32 per minute, the pulse rate 110 per minute and the temperature 99° F. She has frequent spasms of silent coughing during which she becomes greatly distressed. The expulsion of some mucus from the larynx is followed by some relief. Speech is clear but difficult owing to breathlessness.

Cranial nerves.—Visual acuity is good. The discs and retinæ are healthy. The pupils are equal, central, circular and regular in outline. They react briskly to both direct and consensual light, and on accommodation. The external ocular movements are full. There is fine horizontal nystagmus on looking both to right and left, the quick component being in the direction of gaze.

The corneal reflexes are brisk and equal. No sensory loss to pin prick over the face was discovered. There is no facial weakness or palatal palsy. The sterno-mastoids act with respiration, but are weak. The trapezii are very weak. The tongue is protruded straight and shows no atrophy or tremor.

Motor functions.—The patient is utterly helpless. The upper and lower extremities are flaccid and motionless. The only movement possible is in the fingers of the right hand, and this is very slight. All the muscles are somewhat atrophied and this is particularly apparent in the small muscles of the hands.

The muscles of the trunk and neck are also largely paralysed. Her neck unless supported falls forward. With each inspiration the head moves forward with the contraction of the sterno-mastoids. No fibrillation was observed.

Reflexes.—The biceps, triceps, supinator, knee and ankle jerks are all quite absent. The plantar responses are both flexor. There was no incontinence of urine or fæces while in hospital.

Sensory functions.—Sensibility to pin-prick was tested without eliciting any peripheral or other loss. Pressure on the calf and hand muscles causes great pain. The soles of the feet are very sensitive.

No trophic changes are apparent.

Examination of the heart shows no abnormality. Numerous moist râles are heard all over both lungs.

Progress.—Her breathing became more and more embarrassed. Abundant frothy expectoration was brought up with great difficulty and exhaustion. She died two days after admission, on October 13, 1929.

Post-mortem Examination by Dr. Greenfield twenty-four hours after Death.

The body was well-nourished but some wasting of the fore-arms was evident. The small muscles of the hands were soft but not obviously atrophic.

The brain and spinal cord were removed. The brain was quite normal in appearance. The cord in the upper dorsal region appeared rather softened. The anterior and to a less extent the posterior roots of both the cervical and lumbar enlargements were greyish but not atrophied.

The nerves in the arms were definitely larger than normal throughout their extent from the brachial plexus downwards and were pinkish in colour. The sciatic nerve was also pinkish but was rather smaller than normal, though possibly more rounded in shape. Pieces of the ulnar, median, sciatic, musculo-spiral, internal cutaneous nerves and brachial plexus were taken for further examination.

Several of the dorsal root ganglia from the cervical, thoracic and lumbar regions were removed with the cord. Pieces of the biceps brachialis, biceps femoris, erector spinæ and diaphragm were kept for examination.

Thorax and abdomen.—The *right lung* was partially collapsed in its lower lobe, so that fairly large pieces sank in water. Apart from evidence of early broncho-pneumonia in the collapsed areas the lungs elsewhere appeared healthy. Microscopic examination of the right lower lobe revealed a large area of collapse surrounded by extensive broncho-pneumonia and much œdema.

The *heart* showed nothing abnormal macroscopically. The *thymus* macroscopically was definite. It was firm and pinkish in colour. Microscopically a large amount of thymic tissue remained but nothing abnormal was recognized.

In the *liver*, macroscopically, nothing abnormal was noted. Microscopically a considerable amount of congestion was present and some fatty change

throughout the lobules. A little cellular proliferation was evident around the portal spaces. There was no evidence of cirrhosis.

The *spleen* macroscopically was normal apart from evidence of mild venous congestion. The *kidneys*, macroscopically, showed only some passive congestion. Microscopically no other abnormality was noted.

The *suprarenals* were merely thin-walled empty capsules.

Examination of the Nervous System and Muscles.

Peripheral Nerves.—Technique: After ten days' fixation in 10 per cent. formalin, pieces of the median, ulnar, musculospiral, sciatic, internal cutaneous nerves and brachial plexus were imbedded in celloidin. Sections of these nerves were also cut on the freezing microtome. Longitudinal and transverse sections were stained with hæmatoxylin and van Gieson, hæmatoxylin and eosin, Scharlach R., toluidin blue and by the Weigert-Pal method.

Microscopic findings.—On examination the nerves all showed the same changes, differing only in degree. Longitudinal sections of the median, ulnar, musculospiral, sciatic and brachial plexus revealed extensive areas of myelin degeneration. The myelin was seen to be beaded and irregular in many places and occasionally it lay between the nerve-fibres in "balls." It was not infrequently absent or almost absent over quite large areas.

Transverse sections of these nerves stained to show myelin illustrated even more strikingly the amount of degeneration. In the median nerve, which was the most severely affected, the majority of the axis cylinders were entirely deprived of myelin and many of the remainder had an abnormally small amount. The funiculi, however, were not all affected equally; some had comparatively little myelin loss, whereas in others scarcely any myelin sheaths remained. The outer cord of the brachial plexus showed almost as advanced a degree of myelin degeneration as the median nerve. The posterior cord of the plexus, the ulnar and musculospiral nerves were less severely affected and the large majority of axis cylinders in these nerves were surrounded by a myelin sheath; in many instances, however, the sheath consisted of an irregular and frequently incomplete ring. In the sciatic nerve also a large proportion of the fibres were deprived of myelin or possessed an inadequate amount. This nerve also demonstrated well that the brunt of the degenerative process tended to fall on certain bundles, leaving others relatively little damaged. (Figs. 1 and 2.)

The internal cutaneous nerve was the only purely sensory nerve examined. In it the myelin degeneration was considerable but much less than in the motor and mixed nerves.

The other changes observed in the nerves were roughly proportional in degree to the amount of myelin loss present. They consisted of a moderate increase in the connective tissue between the nerve fibres, but this was not sufficient to account for the increase in size of the nerves noted at post-mortem examination. Proliferation of the sheath of Schwann cells was a conspicuous feature in all the nerves, and the nuclei were, in general, larger and more circular in shape than is normally the case.

PLATE XI.

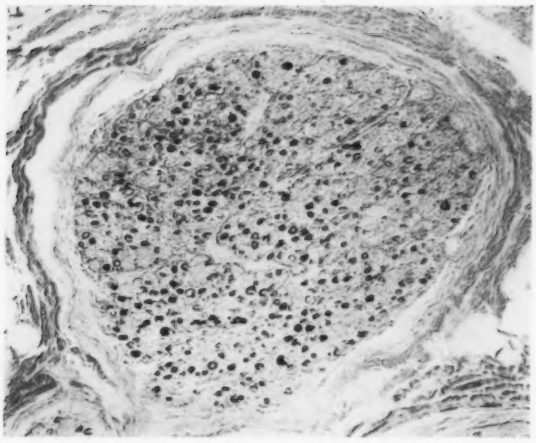

FIG. 1.—Sciatic nerve—high power. Shows advanced demyelination in a nerve bundle. Weigert-Pal (celloidin section).

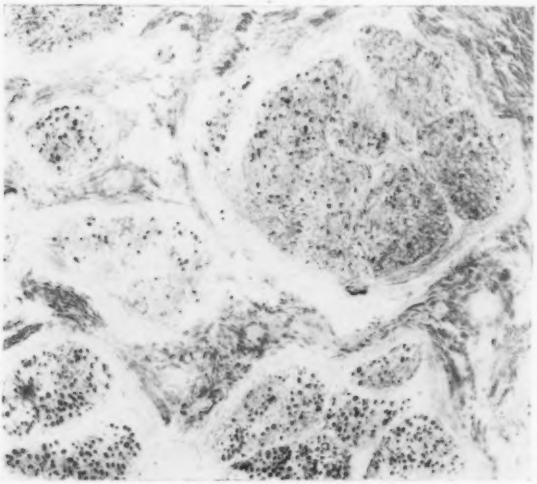

FIG. 2.—Median nerve—low power. Shows severe myelin loss, much more pronounced in some funiculi than in others. Weigert-Pal (celloidin section).

To illustrate paper by Herbert H. Hyland and W. Ritchie Russell.

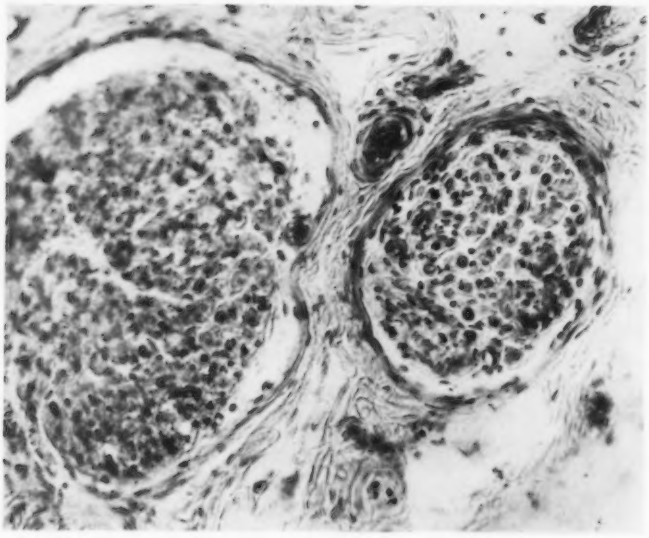

FIG. 3.—Two intramuscular nerves in the biceps femoris—high power. Myelin degeneration is advanced. The dilated perineural space is seen in both nerves, but is more prominent in the larger. Weigert-Pal (celloidin section).

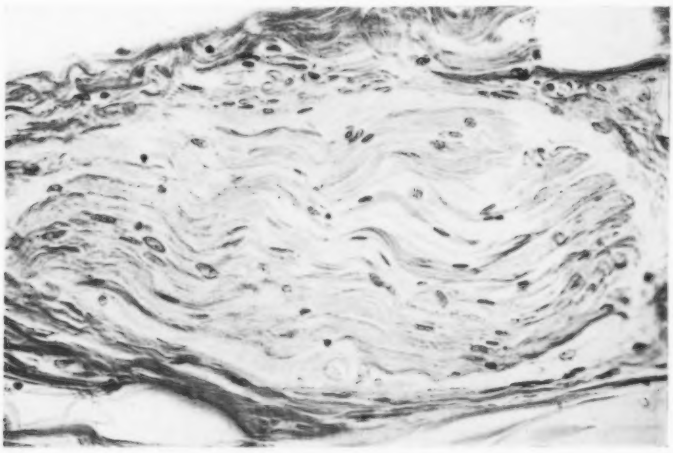

FIG. 4.—Small nerve in the biceps brachialis—high power. The dilated perineural lymph space is well seen. The fibres are seen to be separated from each other, suggesting œdema within the nerve. Hæmatoxylin and van Gieson (celloidin section).

To illustrate paper by Herbert H. Hyland and W. Ritchie Russell.

PLATE XIII.

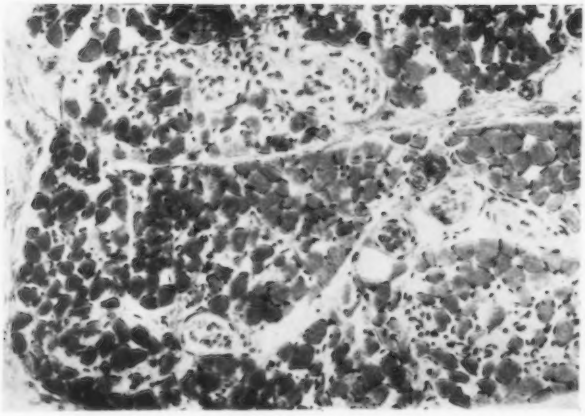

FIG. 5.

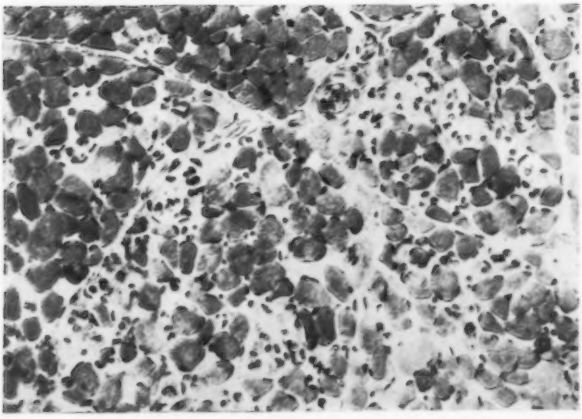

FIG. 6.

FIGS. 5 and 6. — Biceps brachialis showing many small nerves grouped amongst atrophied muscle fibres. × 100. The nerves appear to be very cellular owing to the presence of endothelial cells in the nerve bundles. Around some of the nerves dilated perineural lymphatic spaces are evident. Hæmatoxylin and van Gieson (celloidin section).

To illustrate paper by Herbert H. Hyland and W. Ritchie Russell.

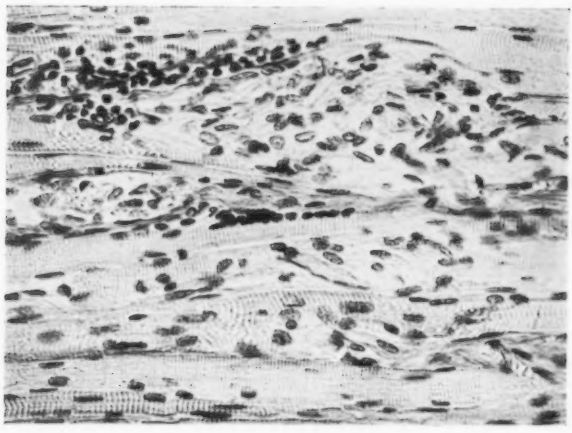

FIG. 7.—Small nerves in longitudinal section of biceps brachialis. × 200. Shows endothelial cells among the nerve fibres and infiltration with small mononuclear cells adjacent to the nerve. Hæmatoxylin and van Gieson (celloidin section).

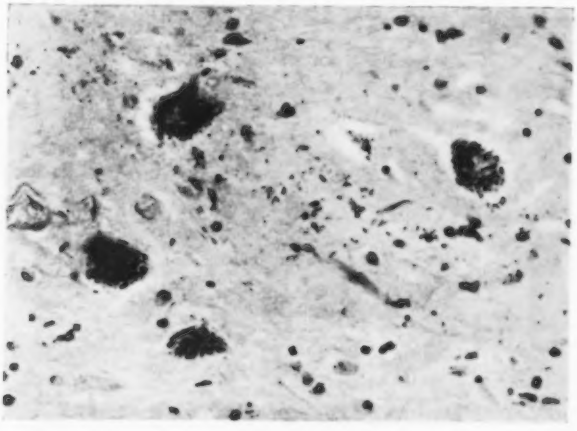

FIG. 8.—Eighth thoracic segment—high power. Four cells from the antero-mesial group of the anterior horn. Shows the roundness of the cells, the tendency to shrink and the variation in size of the Nissl granules, many of which are very large. The Nissl granules tend to be grouped more towards the centre of the cells, leaving a narrow, clear area at the periphery. The nuclei are central. Nissl (celloidin section).

To illustrate paper by Herbert H. Hyland and W. Ritchie Russell.

In addition to the proliferative changes there was an extensive infiltration by mononuclear cells throughout the nerves. Most of these cells were small lymphocytes, but a number of endothelial wandering cells was present and also a few plasma cells. No polymorphonuclear cells were seen. The infiltration was principally between the nerve bundles in the connective tissue spaces, tending to be more dense in the vicinity of the blood-vessels; but in smaller degree it was scattered diffusely amongst the nerve-fibres as well.

The large clear nuclei of lymphoblasts were present around some of the blood-vessels, not infrequently forming several layers.

A great increase of vascularity was observed in all the nerves. No definite thickening of the vessel coats was present.

In sections stained with hæmatoxylin and eosin many cells were seen which at first glance resembled plasma cells. The cytoplasm, however, was slightly granular and was pink with a rose tint; is was also less extensive than in the plasma cell. The chromatin in the nuclei, although tending to have a peripheral distribution, was not so heavy as in the plasma cell. These cells were recognized to be tissue mast cells, and when stained with toluidin blue or methyl violet showed the characteristic granules.

In sections stained with toluidin blue the sheath of Schwann nuclei were seen, in many instances, to be surrounded by π granules.

Comparison was made between the nerves from this case and eleven nerves considered normal, to determine if the number and distribution of mast cells bore any relation to the pathological process present.

A number of π granules as great as that in the nerves under examination was present in nine of the normal nerves.

The number of mast cells in four of the normal nerves was considerably less than that found in the nerves of this case, but in five of the normal nerves it was just as great, and in the remaining two there were more mast cells to be seen than were present in any of the pathological nerves.

The mast cells in the normal nerves appeared to have a similar distribution to those in this case, lying for the most part amongst the connective tissue fibres and in the neighbourhood of blood-vessels.

It was concluded from this investigation that the number of π granules and mast cells in the nerves of this case were without pathological significance.

INTRAMUSCULAR NERVES.—Technique: After fixation for ten days in 10 per cent. formalin saline pieces of the biceps brachialis, biceps femoris, erector spinæ and diaphragm were embedded in celloidin and thin sections were cut and stained with hæmatoxylin and van Gieson, Mallory's aniline blue—orange G. stain and by the Weigert-Pal method.

Microscopic findings.—A striking change was seen in relation to the intramuscular nerves in all the muscles examined with the exception of the diaphragm. This was a dilatation of the perineural lymph spaces which appeared as clear zones surrounding the nerve bundles lined both internally and externally by connective tissue and in some places broken by fine strands of connective tissue (figs. 3 to 7). There was no definite evidence of serous

or cellular exudate in these spaces. They were found around nerve bundles of all sizes, including the finest ramifications. The possibility that they might be due to artefact was considered, but was negatived by the regularity and constancy with which they appeared. A similar but very much slighter degree of the same change could be seen in the large peripheral nerves such as the median. It is likely here that the shrinkage of connective tissue fibres inseparable from formalin fixation would have reduced the size of the spaces and so rendered them less apparent.

In sections of these muscles there were areas where very small nerve bundles were numerous and closely grouped together. The nerves in these areas, both in longitudinal and transverse sections, presented a very cellular appearance, owing to the presence of many endothelial cells among the fibres and to a possible increase in connective tissue nuclei. The appearance suggested an actual proliferation probably originating in the cells lining the perineural lymphatics. The fibres in the smaller nerves seen in longitudinal section were frequently widely separated, suggesting the presence of œdema inside the bundles. It is notable that the larger intramuscular nerves showed no infiltrative or proliferative change whatever.

The intramuscular nerves of the diaphragm did not possess the dilated perineural lymph spaces, nor was there any proliferative change in the nerves of this muscle.

Transverse sections of the muscles stained by the Weigert-Pal method showed the intramuscular nerves to be deprived of their myelin in varying degree. Many of the nerves in all the muscles except the diaphragm contained a large number of the fibres entirely without myelin. However, considerable variation existed in the individual muscles, some of the nerves having little less than the normal amount, whereas certain bundles were almost entirely demyelinated. In longitudinal sections beading and irregularity of the myelin were very pronounced in most of the nerves.

The most severe degeneration was seen in the nerves of the biceps brachialis. Those in the biceps femoris were a little less affected and the nerves in the erector spinæ had less myelin loss than the two former muscles, although in many of the nerves it was quite severe. The nerves of the diaphragm were well myelinated, although here also some beading and irregularity were observed.

The sensory nerves in the muscle spindles were, for the most part, well myelinated. In a few instances the myelin was absent, but in general it was far better preserved in these fibres than in the motor nerves.

Around some of the muscle spindles dilated spaces were seen similar to those surrounding the motor intramuscular nerves.

SPINAL CORD.—Technique: After ten days' fixation in 10 per cent. formol saline, sections were cut at various levels on the freezing microtome and stained by the Marchi method and Scharlach R.

Pieces from every segment of the cord were embedded in celloidin and sections cut from each segment were stained by the following methods: Loyez's stain for myelin, hæmatoxylin and van Gieson, toluidin blue. Sections from every alternate segment were stained by the Weigert-Pal method.

Microscopic examination.—Despite careful examination, no degeneration of the white matter was observed in the cord. The cells of the anterior horns, however, varied definitely from the normal shape and presented a rounded appearance throughout the cervical, thoracic and lumbar segments. Many cells in the cervical and thoracic regions were so shrunken in size that at first glance the cells were thought to be diminished in number in all the cell groups. A count of the cells showed that the actual diminution in number was slight, though in places many of the cells were so small as to be only just recognizable as such. The segments possessing the largest numbers of shrunken cells were C. 7, Th. 3, 6, 7, 8 and 9. In the remaining cervical and thoracic segments the cells were considerably nearer to the normal in size, and in the lumbar and sacral regions there was practically no evidence of shrinkage.

The nuclei of the cells were normally situated and no true chromatolysis was present. The Nissl granules, however, were large and irregular in size in the rounded cells and in many instances tended to clump in the centres of the cells, leaving a narrow clear area of cytoplasm at the periphery (fig. 8). These changes in the Nissl granules were most obvious in the thoracic segments, but they were also present in varying degree in segments C. 3, 6, 7 and 8 and L. 1 and 2. No abnormality of the Nissl granules was seen in the remainder of the cord. No excess of normal yellow pigment was present in the cells. The cells of Clarke's column were normal.

A great increase in vascularity was present throughout the grey matter. The vessels were much more numerous than is normally the case and were dilated and engorged with blood corpuscles. The walls of many of the vessels were thickened by an increase of connective tissue, but no proliferation of the intimal lining was present.

DORSAL ROOT GANGLIA.—Technique : After fixation with the cord, ganglia from the cervical, thoracic and lumbar regions were imbedded in paraffin and sections were stained with hæmatoxylin and van Gieson, toluidin blue and Mallory's anilin blue-Orange G. stain.

Microscopic findings.—No abnormality was observed in the size or shape of the cells. The nuclei were centrally situated and no chromatolysis was present. There was a definite thickening of the capsules surrounding many of the cells due to a proliferation of capsule cells. This change was most apparent in the ganglia from the thoracic region.

ANTERIOR AND POSTERIOR NERVE ROOTS.—Technique : The same technique was employed as for the spinal cord.

Microscopic findings.—Hæmatoxylin and van Gieson-stained sections revealed little, if anything, abnormal in the posterior roots except an increase in vascularity. In the anterior roots the increase in vascularity was greater and a few mononuclear cells were seen amongst the nerve fibres. No advanced proliferative or infiltrative changes were present in the nerve roots such as were seen in the peripheral nerves.

Weigert-Pal sections presented a striking picture. The posterior roots were all normally myelinated except in one or two of the thoracic segments where

slight loss was evident in portions of the roots at some distance from the cord. The anterior roots, however, had extensive myelin loss throughout the lower cervical, thoracic and upper lumbar regions of the cord. This varied in degree in different segments but it was well marked in all, and in a number of the lower cervical and thoracic segments the proportion of demyelinated fibres equalled that found in the peripheral nerves (fig. 9).

In the roots of the cauda equina no infiltrative or proliferative changes were present. No myelin degeneration was seen in these roots stained by the Marchi method.

BRAIN.—Technique: After ten days' fixation in 10 per cent. formalin saline frozen sections were cut from the prefrontal lobe and the precentral and post-central convolutions. The sections were stained with Scharlach R., toluidin blue and Bielschowsky's stain for neurofibrils.

Microscopic findings.—The sections revealed no abnormality in the cells or neurofibrils and no increase in the deposition of fat.

MEDULLA.—Technique: After fixation with the brain the medulla was imbedded in celloidin and serial sections were cut through its entire length. Every tenth section was stained with hæmatoxylin and van Gieson and toluidin blue.

Microscopic findings.—No abnormality was present in any of the cell groups. The cells of the dorsal nucleus of the vagus and the nucleus ambiguus were normal in size and appearance. Great increase in vascularity was evident throughout the medulla; the vessels were extremely numerous and engorged with blood.

The greatest increase in vascularity existed in the region occupied by the dorsal nucleus of the vagus, and in two places, at considerable distance apart, moderately large, fresh hæmorrhages were seen amongst the cells of this nucleus.

A slight perivascular infiltration of mononuclear cells existed throughout the medulla.

MUSCLES.—*Technique*: The methods used for the muscles was described when the intramuscular nerves were considered.

Microscopic findings.—Measurement of the fibres and comparison with normal muscles revealed that the fibres of the biceps brachialis, biceps femoris and erector spinæ were very much reduced in size. The great majority of the fibres in the biceps brachialis were between 15 and 30 μ in diameter; in the other two muscles they averaged a little larger. All three muscles contained many very small fibres however, and fibres 5 to 10 μ in diameter were frequently seen. Occasionally amongst the atrophied fibres were little groups of large fibres varying in size from 60 to 110 μ in their smallest diameter, but these were not common in the muscles. The fibres of the diaphragm averaged little if at all less than the normal size; no gross irregularity in size or shape was evident.

The fibres in all the muscles were well striated, and apart from atrophy no other abnormality was observed. A moderate increase of connective tissue was seen in all the muscles except the diaphragm. There was no increase in the sarcolemma nuclei.

PLATE XV.

FIG. 9.—Ninth thoracic segment—low power. The posterior roots are well myelinated, whereas advanced degeneration is present in both anterior roots. Weigert-Pal (celloidin section).

To illustrate paper by Herbert H. Hyland and W. Ritchie Russell

The diaphragm was extensively infiltrated with little groups of small round cells. These usually surrounded small blood-vessels lying between the muscle fibres. A very much less extensive infiltration was present in the other three muscles. Small groups of mononuclear cells were seen in the connective tissue spaces or between the muscle fibres, having no relation to blood-vessels. Occasionally a group of these cells was adjacent to one of the small nerves in which endothelial proliferation was occurring.

Summary of Pathological Findings.

The body of a well-nourished woman with no gross atrophy except moderate wasting of the fore-arms. The nerves in the upper limbs were definitely larger than normal and the cord in the thoracic region appeared rather softened. Apart from broncho-pneumonia and partial collapse of the right lung no other changes of importance were noted outside the nervous system.

Microscopic examination of the nervous system revealed that the anterior horn cells in the spinal cord were rounded and shrunken. The Nissl granules were pyknomorphic and had a tendency to clump in the centres of the cells. No actual chromatolysis was present and there was no degeneration of the white matter in the cord.

The anterior nerve roots, the peripheral nerves and the intramuscular nerves were severely demyelinated. The posterior nerve roots were very slightly affected in this respect and the sensory nerves in the muscle spindles had relatively less myelin loss than the motor intramuscular nerves. The only purely sensory nerve examined, the internal cutaneous, had less myelin degeneration present than the other peripheral nerves.

All the peripheral nerves showed proliferation of the interstitial tissue and were infiltrated by mononuclear cells.

Two interesting findings in the intramuscular nerves of the muscles of the back and limbs were a dilatation of the perineural lymph spaces and a proliferation of endothelial cells inside the small nerve bundles where the latter lay in groups between the muscle fibres.

Many of the cell capsules of the dorsal root ganglia were definitely thickened by proliferation of the capsule cells.

Nothing abnormal was found in the brain and the medulla was normal apart from great increase of vascularity and two recent hæmorrhages amongst the cells of the dorsal nucleus of the vagus.

In the muscles of the limbs and back there was severe generalized atrophy of the fibres. The diaphragm was not atrophied but extensive perivascular infiltration by mononuclear cells was present.

Commentary.—Polyneuritis arising from various causes may progress slowly, but the great majority of cases show remissions or become arrested before paralysis is extreme. A clinical course similar to that in the case described is particularly unusual. The type is, however, probably identical with that described by Harris [4], although his cases recovered from the extreme paralysis which occurred. The fatal termination in this case provided a valued opportunity for one of us (H.H.H.) to investigate its pathology.

Various changes have been described in the spinal cords of cases of peripheral neuritis. In the white matter the posterior columns are most frequently affected, as has been emphasized by Herzog [5] and Cole [2]. The latter, indeed, after an extensive search of the literature up to 1902, found no record of a case in which the cord failed to show degeneration of these columns if the Marchi method had been employed.

Where changes have been found in the anterior horn cells, they have usually consisted of a varying degree of central chromatolysis with displacement of the nuclei to the periphery of the cell.

Grinker, in 1907 [3], expressed the view that cases of polyneuritis usually showed no changes either in the cord or anterior roots. Pfeifer [7], however, more recently re-emphasizes the frequency with which changes are found in both white and grey columns of the cord and also in the brain.

It should be noted that very different types of polyneuritis have been included in these observations. Grinker's case resembled in many ways the case here described, especially in its clinical course, but the pathological examination (which was incomplete) showed no cord changes.

In the present case an exhaustive search revealed no trace of degeneration in the white matter of the cord, nor were there any changes detected in the brain. In the grey matter the changes found in the anterior horn cells resembled in many respects those found in progressive muscular atrophy. The changes in the Nissl granules presented a somewhat similar picture to those described by Martin [6] in cases of syphilitic progressive muscular atrophy.

The changes in the peripheral nerves were similar to those found in other types of severe polyneuritis. The predominance of motor as compared to sensory nerve involvement was prominent both clinically and pathologically and corresponded to the clinical findings in Harris's cases.

As regards the ætiology of this type of neuritis, Harris suggests that the slow development in such cases indicates a continuously acting toxin

rather than an acute poisoning, and that one is driven to the supposition that an auto-toxæmia is at work. In his second case the absorption of external toxins could probably be excluded, as the patient continued to get weaker for nine months after returning from South Africa to England. In the case here considered, this factor could probably also be excluded as her downward progress was uninterrupted during a stay of seven weeks in hospital.

Cases have occurred following acute illnesses, such as that described by Barnes (*vide supra*) which followed rheumatic fever. It is noteworthy that both one of Harris's cases and the present case developed after a dermatitis of doubtful nature.

The dilatation of perineural lymphatic spaces which was an interesting factor in this case was also noted by Stewart [9] in a case of fatal puerperal polyneuritis and poliomyelitis. In his case the changes were found in the large nerve trunks, whereas in this case they were most marked in the intramuscular branches.

We wish to express our thanks to Dr. James Collier and to Dr. Russell Brain for permission to publish the case; and to acknowledge our indebtedness to Dr. J. Godwin Greenfield for his assistance with the pathological investigation and for taking the photomicrographs.

Summary.

(1) A case of polyneuritis which ran a progressive course and terminated fatally in twelve months is reported.

(2) Somewhat similar cases are referred to.

(3) A pathological study of the case is described (see Summary of Pathological Findings).

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VASCULAR LESIONS OF THE HIND-BRAIN. (LATERAL MEDULLARY SYNDROME.)[1]

BY HOUSTON MERRITT, M.D.
AND
MAXWELL FINLAND, M.D.

VASCULAR lesions of the lateral portion of the medulla are generally known as the syndrome of the occlusion of the posterior inferior cerebellar artery. This syndrome was first described by Wallenberg [1] in 1895, although very similar cases were reported by Senator [2] eleven years previously. Since the first description there have been a limited number of cases reported, chiefly in the French literature. Xavier Combes [3] in 1902 reviewed eight cases of the syndrome, including the cases reported by Babinski and Nageotte, Cestan and Hoffman, Foix, Hillemand and Schalit [4] in 1925 reported a case and analysed sixteen previously reported cases from the standpoint of the blood-vessel involved. Their conclusions will be mentioned later. In the American literature the subject has received attention from H. M. Thomas [5] in 1907, W. G. Spiller [6] in 1908, G. W. Robinson [7] in 1913, R. H. Thompson [8] in 1929, and W. H. Riley [9], in 1930. In the English literature cases have been reported by Menzies [10] in 1893, S. A. K. Wilson [11] in 1909, Wilfred Harris [12] in 1910, and Ramsbottom and Stopford [13] in 1924.

The syndrome is supposed to be comparatively rare, but at the Boston City Hospital we have met six cases in the past two years, and Riley [9] of Battle Creek, Michigan, encountered eight cases in a similar period. It is probable, therefore, that the syndrome is fairly common.

The following six cases were observed at the Boston City Hospital during the years 1928 and 1930. As far as we are able to determine four of these patients are still living. Two of the patients died while in the hospital, and post-mortem examination was made on one of these.

[1] Read before the Boston Society of Psychiatry and Neurology, February 20, 1930. From the Second Medical and Neurological Services of the Boston City Hospital, and the Department of Neuropathology, Harvard Medical School, Boston, Mass.

A summary of the history and abnormal findings in these cases is as follows :—

Case 1.—James L., aged 69, retired salesman, admitted December 15, 1927, to the Second Medical Service of the Boston City Hospital. He died March 15, 1928. His chief complaints were vertigo and dysphagia.

Family history and past history were not important except that he had a "stroke" in 1913, resulting in a paralysis of the left arm and leg. He recovered rapidly without any residual paralysis.

Present illness.—On the morning of December 14, the patient became nauseated and vomited, and felt very dizzy. He did not lose consciousness, however, and was helped into a taxi-cab. He went home and went to bed. He then began to notice a sharp pain in the central portion of the right half of the face. The dizziness persisted and he vomited several times more. In the afternoon he found that he could not swallow, and that foods and liquids regurgitated through his nose. He also noticed diplopia. On the advice of his physician he presented himself at the hospital the following day and was admitted.

Physical examination.—The peripheral vessels were moderately sclerosed and his blood-pressure was 164/100.

Neurological examination on admission showed the following abnormalities (see fig. 1) :—

Cranial nerves :—

(1) There was a mild Horner's syndrome on the right (i.e., enophthalmos, myosis, and drooping of the lid with narrowing of the palpebral fissure).

(2) The eye movements were normal, but there was a well-sustained horizontal nystagmus, more marked on right lateral deviation.

(3) There was impairment of light touch and pain sensation in the distribution of the right trigeminal nerve.

(4) There was definite, although slight, weakness of the right half of the face.

(5) His voice was hoarse, and swallowing was very difficult. The palate deviated to the left on phonation.

Motor system.—No disturbance of the motor system could be detected.

Sensory system.—There was impairment of sensation to light touch and pain in the left arm and leg. Other forms of sensation were not tested.

Reflexes.—The deep reflexes were more active on the left side. The plantar responses were normal. The abdominal reflexes were absent.

Laboratory data.—The urine and blood were normal. The blood Wassermann test was negative.

Course in the hospital.—The sensation of dizziness cleared rapidly and the dysphagia disappeared in the course of a month. The hoarseness persisted for about ten weeks. The Horner's syndrome was much less noticeable after a few weeks but persisted until death. During the first few days after admisssion the patient was troubled with intractable hiccoughing. Eleven weeks

after admission the patient developed erysipelas on the right side of the face, and died eight days later.

The *pathological findings* at necropsy were broncho-pneumonia, erysipelas, marked arterio-sclerosis, and chronic vascular nephritis. The brain (N-28-9) showed marked sclerosis of the basilar vessels, but no detailed study of them was made. The cerebrum appeared normal on gross examination. The medulla was sectioned serially.

The sections of the medulla showed a mild degeneration of the pyramidal tract on the right side. This extended through all the sections, and was separated from the lesion in the lateral portion of the medulla; it was con-

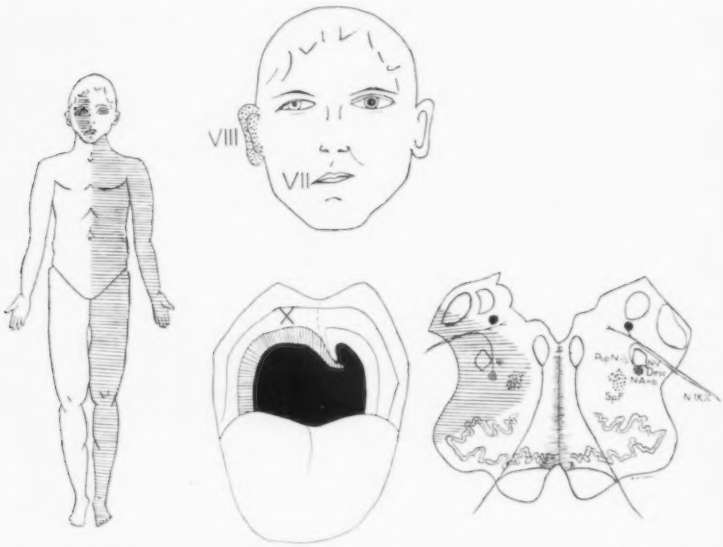

FIG. 1 (*Case* 1).—Sketch showing cranial nerves injured, the distribution of the sensory changes and the approximate location of the lesion in the medulla.

sidered to be the result of the "stroke" in 1913. The interesting lesion, however, was a triangular area of degeneration in the lateral portion of the medulla extending from just above the cuneate and gracile nuclei to the level of the entrance of the restiform body into the cerebellum. It involved the lateral reticular substance and destroyed the nucleus ambiguus, the oculo-pupillary nucleus, the spinal lemniscus, the descending nucleus and tract of the trigeminal nerve, and the lateral reticular nucleus. In the lower sections there was a slight involvement of the extreme lateral portion of the restiform body. A small portion of the dorso-lateral edge of the olivary nucleus was also destroyed. The hypoglossal nerve nucleus and the medial lemniscus were not involved. The lower end of the tractus solitarius was destroyed.

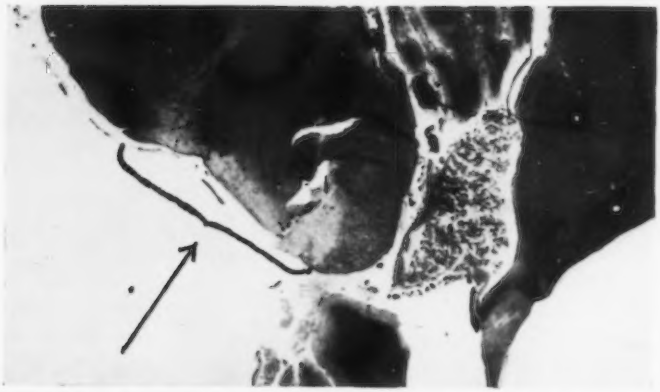

FIG. 2 (*Case* 1).—Photograph of section of the right side of the medulla showing the lower level of the lesion. The degenerated area lies chiefly in the restiform body and in the lateral reticular substance. Weigert's stain.

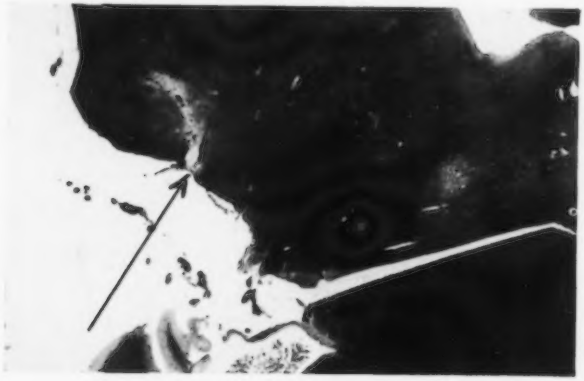

FIG. 3 (*Case* 1).—Photograph of section of the right side of the medulla showing the central portion of the lesion. The area of degeneration is confined to the lateral reticular substance. Weigert's stain.

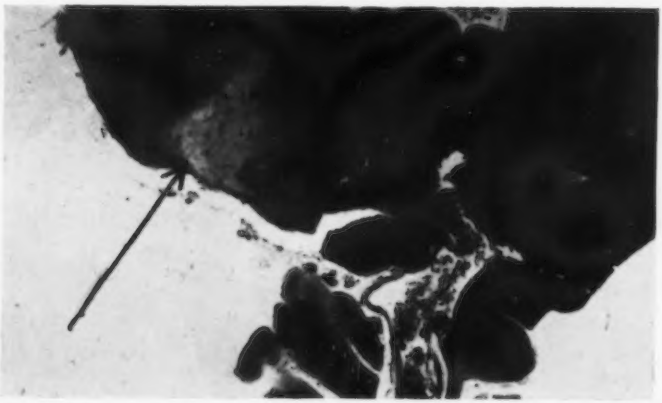

FIG. 4 (*Case* 1).—Photograph of section of the right side of the medulla showing the upper level of the lesion. The area of degeneration lies in the lateral reticular substance and involves the dorsal portion of the olivary nucleus. Weigert's stain.

To illustrate paper by Houston Merritt and Maxwell Finland.

Figs. 2, 3 and 4 show photographs of sections taken from the lower, central and upper levels of the lesions, respectively.

Case 2.—Estelle W., aged 69, widow, unemployed, admitted to the Second Medical Service of the Boston City Hospital October 10, 1927, and discharged April 29, 1928, as improved.

The patient had been receiving treatment in the out-patient department of the hospital for a period of about seven years on account of chronic nephritis and hypertension. On October 10, 1927, she was accidentally overcome by coal gas, and was brought to the hospital. She recovered rapidly from the gas poisoning, but remained in the hospital for treatment of her nephritis.

On January 14, 1928, while still in the hospital, she complained of a severe pain over left side of her face, and also of marked dizziness, and diplopia. These symptoms persisted, and the following day herpetic lesions appeared on the lips, and she became unable to swallow, food regurgitating through her nose. Her voice became husky and she had intractable hiccoughing. She complained of pain in the left side of her throat, and a cold sensation with numbness on the left side of face and right side of the body.

Physical examination.—The heart was moderately enlarged, the peripheral vessels were sclerotic and the blood-pressure was 260/100.

Neurological examination on January 16, 1928, showed the following abnormalities (see fig. 5):—

Cranial nerves:—

(1) There was a left-sided Horner's syndrome.

(2) The eye movements were of normal range, but the patient complained of diplopia on looking to the left.

(3) There was an impairment of the light touch and pain sensibility on the left side of the face.

(4) The voice was husky and there was marked dysphagia. The palate deviated to the right on phonation.

There was marked dizziness, but no nystagmus or impairment of hearing was noted.

Motor system.—There was a very slight weakness of the right arm and leg.

Sensory system.—There was hypæsthesia (modalities not noted) of the skin of the right arm and leg with a subjective sensation of numbness and tingling.

Reflexes.—The deep reflexes were more active on the right side than on the left. The abdominal and plantar reflexes were normal.

Laboratory data.—The urine showed the changes found in chronic nephritis. The blood-count was normal, and the blood Wassermann was negative.

Course in the hospital.—Patient was discharged from the hospital about three and a half months after the vascular accident. The hoarseness had disappeared, and there was only slight dysphagia. There was still slight weakness of the left half of the face and of the right arm and leg. The

subjective numbness and objective sensory changes persisted. Horner's syndrome had disappeared.

Case 3.—Mary J., aged 61, housewife, admitted to the First Medical Service of the Boston City Hospital, November 18, 1928. She was discharged December 23, 1928, as improved. Her chief complaints were dizziness, dysphagia and hoarseness.

Family history and past history were irrelevant.

Present illness.—The patient had been under treatment for hypertension, and on November 17 she went to her physician to consult him about constipa-

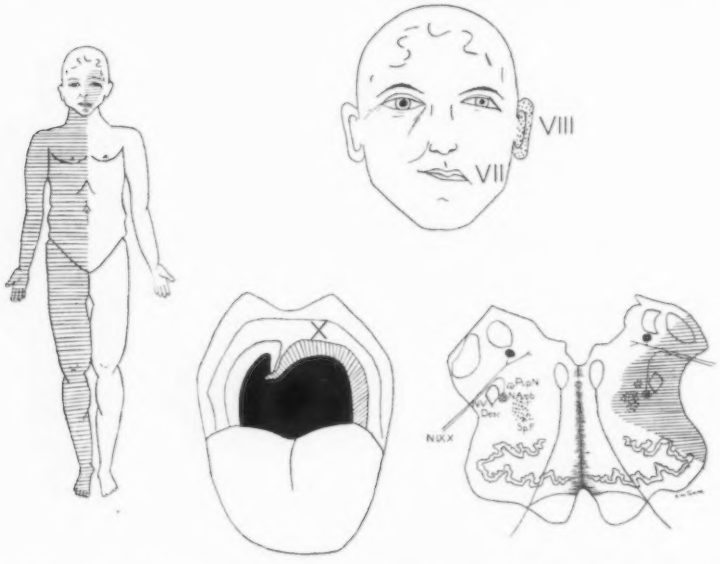

FIG. 5 (*Cases* 2 and 3).—Sketch showing the cranial nerves injured, the distribution of the sensory changes, and the approximate location of the lesion in the medulla.

tion. While there she suddenly became unconscious and fell to the floor. She regained consciousness in about twenty minutes and noted a pain in the left side of her head and a sensation of coldness and numbness in the right arm and leg. Her voice was husky and she had great difficulty in swallowing, liquids and solids regurgitating through her nose. These symptoms persisted and she was permitted to the hospital on the following day.

Physical examination.—The heart was enlarged and was irregular in rhythm owing to premature systoles. The peripheral vessels were sclerosed and the blood-pressure was 180/100. The blood and urine were normal and the blood Wassermann reaction was negative.

Neurological examination on admission showed the following abnormalities (see fig. 5):—

Cranial nerves:—

(1) Horner's syndrome on the left side.

(2) There was limitation of movement of the eyes to the extreme left and patient complained of diplopia on looking to the left.

(3) There was impairment of sensation to pain and temperature in the distribution of the left trigeminal nerve. The left corneal reflex was absent and there was a very slight weakness of the masticatory muscles on the left side.

(4) There was a slight weakness of the left half of the face involving both upper and lower portions.

(5) There was horizontal nystagmus, subjective vertigo and lateropulsion to the left.

(6) The voice was husky. There was marked dysphagia and the palate deviated to the right on phonation.

The motor system was normal.

Sensory system.—There was impairment of sensation to pain and temperature in the skin of the right arm, trunk and leg. Light touch, vibratory and position sense were normal. There was a subjective sensation of numbness of the right arm, trunk and leg.

Reflexes.—The deep reflexes and the plantar responses were normal. The abdominal reflexes were absent on the left side.

Course in the hospital.—The patient remained in the hospital for five weeks. At the end of this time she was able to swallow without difficulty and her voice was much less husky. The subjective vertigo and the lateropulsion had disappeared. The Horner's syndrome and the sensory changes were still present, however.

Case 4.—Catherine S., aged 75, unmarried and unemployed, admitted November 3, 1929, to the First Medical Service of the Boston City Hospital. She died November 13, 1929.

Her chief complaints were difficulty in swallowing, dizziness and sudden weakness.

Family history and past history were not important. The patient had ceased work nine years previously, and since that time had remained at home. For the past year or more she had complained of increasing weakness and fatigability.

Present illness.—The patient awoke October 30, 1929, and attempted to get out of bed. She fell back unconscious and remained so about half an hour. On gaining consciousness she was very dizzy and she had great difficulty in swallowing. Her voice was husky. She was brought to the hospital four days later and a connected story of her illness was difficult to obtain as there was a definite impairment of her memory..

Physical examination.—Patient was an obese white woman. The heart

was enlarged and the rate was rapid and irregular. The peripheral vessels were sclerotic and the blood-pressure was 150/100.

Neurological examination on admission showed the following abnormalities (see fig. 6) :—

Cranial nerves :—

(1) There was a Horner's syndrome on the right side.

(2) There was a weakness of the right half of the face involving both the upper and lower portions.

(3) There was slight horizontal nystagmus.

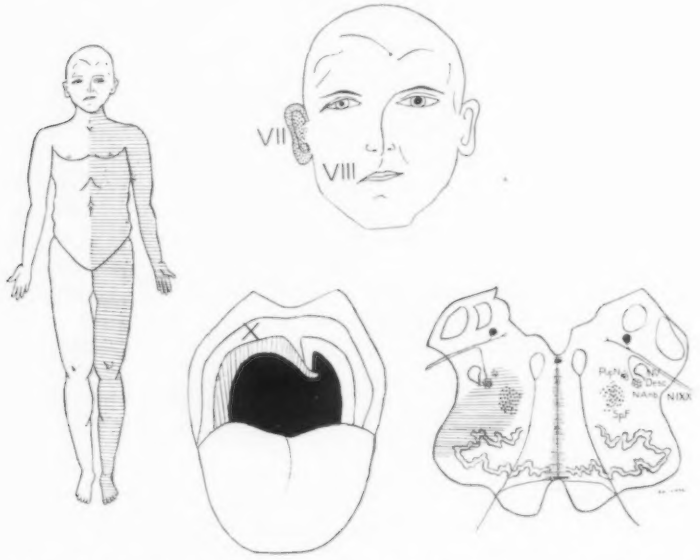

FIG. 6 (*Case* 4).—Sketch showing the cranial nerves injured, the distribution of the sensory changes, and the approximate location of the lesion in the medulla. (In the full face diagram the Roman figures VII and VIII should be transposed.)

(4) The voice was hoarse, and there was marked dysphagia. The palate deviated to the left on phonation.

(5) The right corneal reflex was absent. There was no loss of any of the forms of cutaneous sensation in the face.

Motor system.—There was a marked generalized weakness, equal on the two sides.

Sensory system.—Sensation to pain and temperature was impaired in the left arm, trunk and leg. There was no impairment of the light touch, vibratory or position sense. The patient complained of a sensation of numbness in the left arm and leg.

Reflexes.—All of the deep reflexes were markedly diminished. The abdominal reflexes were absent. The plantar responses were normal.

Laboratory data.—The blood and urine were normal. The blood Wassermann test was negative. The cerebrospinal fluid was under a pressure of 240 mm. of water. It was clear and colourless and contained 5 lymphocytes per cubic mm. The total protein content was 50 mgm. per 100 c.c. The colloidal gold and Wassermann tests of the fluid were negative.

Course in the hospital.—The patient gradually became more feeble, the heart's action weakened and she died on November 13, 1929, ten days after admission.

Case 5.—Annie D., aged 43, housewife, admitted February 1, 1930, to the Third Medical Service of the Boston City Hospital and transferred to the Neurological Service. She was discharged April 13, 1930, as improved.

Her chief complaints were inability to swallow and numbness of the left half of face and body.

Past history and family history were irrelevant.

Present illness.—On January 26, 1930, the patient was confined to bed with a cold. Four days later, she attempted to get out of bed and suddenly became dizzy. The room seemed to be "spinning around," and it was necessary for her to hold on to a table to avoid falling. Her head began to ache and she noticed a ringing in her right ear. On looking to the right objects appeared double. All these symptoms persisted, and about two days later she noticed a numbness and tingling of the left side of her body, beginning in the arm, spreading downward to the leg and foot, and finally involving the left half of the face. Later in the day she became unable to swallow her own saliva and she was sent to the hospital by her physician.

Physical examination.—The heart was enlarged especially to the left, and the typical murmurs of mitral and aortic valve lesions were heard. The rate and rhythm were normal. The blood-pressure was 135/85. The abnormality of the heart was considered as being rheumatic in origin.

Neurological examination on admission showed the following abnormalities (see fig. 7):—

Cranial nerves:—

(1) Horner's syndrome was present on the right side.

(2) The ocular movements were of normal range but the patient complained of diplopia on right lateral deviation.

(3) There was a loss of pain and temperature sensation in the distribution of the right trigeminal nerve. The right corneal reflex was absent.

(4) The right face was slightly weak in both the upper and lower portions

(5) There was slight impairment of hearing of the nerve deafness type in the right ear. There was lateral and vertical nystagmus. Patient complained of a ringing in the right ear, and a dizziness when looking to the right. There was slight lateropulsion to the right.

(6) There was extreme difficulty in swallowing. The palate deviated to the left on phonation.

Motor system.—The right arm and leg were slightly weak and hypotonic. There was intention tremor, inco-ordination, cerebellar rebound and past pointing on the right side.

Sensory system:—

(1) There was a subjective sensation of numbness of the entire left half of body, including face, arm, trunk and leg.

(2) Sensation to pain and temperature was lost in the skin of the left half of

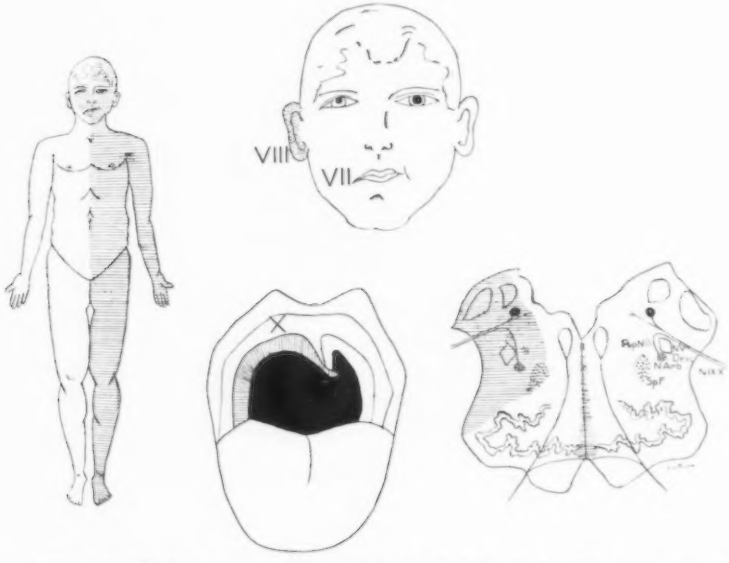

FIG. 7 (*Case* 5).—Sketch showing the cranial nerves injured, the distribution of the sensory changes, and the approximate location of the lesion in the medulla.

the body, including the face. There was loss of the pain and temperature sensibility in the right trigeminal nerve distribution, as noted above. When the left side of the body was touched with a hot or cold object or with a pin the patient complained of a "peculiar sensation," which was disagreeable and uncomfortable, but she was unable to describe it further.

(3) Sensation to light touch (cotton) was present everywhere, but patient stated that she felt it better on the right half of the body.

(4) There was loss of the sense of taste on the right half of the tongue.

(5) Vibration and position sense were normal.

Reflexes.—The left knee-jerk was slightly hyperactive, and the plantar response was absent on the left side. The abdominal reflexes were normal.

Laboratory data.—The blood and urine were normal. The blood Wassermann test was negative. The spinal fluid was under pressure of 150 mm. of water. It was clear and colourless, with no cells. The total protein content was 10 mg. per 100 c.c. The colloidal gold was 1112110000, and the Wassermann test was negative.

Course in the hospital.—The patient improved gradually and when discharged on April 13, 1930, she could swallow liquids and solids without difficulty. The dizziness and cerebellar ataxia had disappeared and she was able to walk well. There was only slight nystagmus on discharge and no weakness of the facial muscles could be detected.

Horner's syndrome was still present and the sensory findings were the same as on admission.

Case 6.—Oscar C., aged 56, negro, engineer, admitted November 18, 1929, to the Neurological Service of the Boston City Hospital, and discharged February 16, 1930, as improved.

His *chief complaint* was inability to talk or swallow.

Family history and past history were irrelevant.

Present illness.—The patient went to work on November 18, 1929, as usual. His helper stated that he seemed quieter than usual and that he was perspiring profusely. He was working with an electric drill and suddenly, without turning it off, he handed it to his helper. He began rubbing the left side of his face and walked outside into the rain. His helper followed and asked what was the difficulty. The patient continued to rub the left side of his face and did not answer. When the question was repeated the patient finally managed to say "I think I have had a stroke." He was brought to the hospital immediately.

Physical examination.—The patient was obese and plethoric. The heart was enlarged to the left and the blood-pressure was 170/100.

Neurological examination.—The following abnormalities were present at time of admission (see fig. 8):—

Cranial nerves:—

(1) There was a Horner's syndrome on the left side.

(2) There was loss of all forms of sensation in the distribution of the left trigeminal nerve. The left corneal reflex was absent, and there was a paralysis of the muscles of mastication on the left.

(3) The left abducens nucleus was paralysed. The patient was unable to look to the left.

(4) There was complete paralysis of the facial muscles on the left side.

(5) There was slight impairment of hearing of the nerve type in the left ear, and there was marked horizontal and vertical nystagmus.

(6) There was moderate dysphagia and extreme dysarthria, his speech being barely intelligible. The palate deviated to the right on phonation.

Motor system.—There was weakness, inco-ordination, ataxia, and intention tremor of the left arm and leg.

Sensory system :—

(1) Patient complained of numbness of the entire right side of his body (face, arm and leg).

(2) There was loss of pain and temperature sensation in right leg, trunk, arm and face.

(3) Light tactile stimuli were readily recognized, but the patient stated they were felt better on the left side than on the right.

(4) Vibration and position sense were normal.

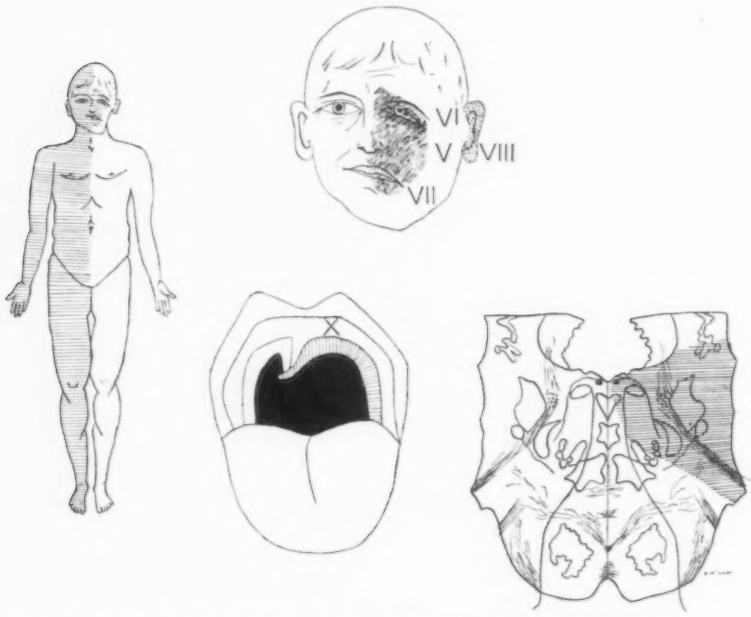

FIG. 8 (*Case 6*).—Sketch showing the cranial nerves involved, the distribution of the sensory changes, and the approximate location of the lesion in the medulla.

Reflexes.—All the deep reflexes were markedly diminished. The right plantar response was equivocal. The abdominal reflexes were normal.

Laboratory data.—The urine and blood were normal. The blood Wasserman test was negative.

The cerebrospinal fluid on the day of admission was under a pressure of 240 mm. of water. The fluid was blood-stained and contained 200,000 red blood-corpuscles and 400 white blood-corpuscles per cubic mm., 80 per cent. of the latter being polymorphonuclear leucocytes, and 20 per cent lymphocytes. The total protein content was 800 mg. per 100 c.c. The colloidal gold test was 0011112233, and the Wassermann test was negative. One week later

the pressure was 200 mm. of water. The fluid was xanthochromic and contained 4,500 red blood-corpuscles, and 555 white blood-corpuscles. The total protein content was 148 mg. per 100 c.c., and the colloidal gold and Wassermann tests were negative.

Course in the hospital —The patient developed a keratitis and pan-ophthalmitis in the left eye which was enucleated on December 16. He was discharged on February 16, 1930, at which time there was obvious atrophy of the masticatory muscles on the left side. The paralysis of the left facial muscles was still complete. The dysarthria had improved markedly, the function of the right internal rectus muscle had returned, and the patient was able to look to the left with the right eye. The patient could walk, but there was still considerable ataxia of the left arm and leg. The sensory changes persisted.

Discussion.

The cases here presented are very similar to those previously reported. The lesion occurs generally in arterio-sclerotic individuals and the age of onset in our cases, as shown in Table I, was in the sixth or later decades, except in Case 5, where the lesion could be explained on basis of an embolus from a rheumatic heart. A few cases have been reported in young individuals, but usually in syphilitics.

The onset is generally fairly sudden without loss of consciousness, and is often followed by a period during which there is an increase in the symptoms.

TABLE I.—Symptoms.

Case	Age and Sex	Time of progress *	Pain in face	Diplopia	Vertigo	Nausea	Dysphagia	Dysarthria	Subjective numbness
1	69 M.	12 hours	+	+	+	+	+	+	0
2	69 F.	24 hours	+	+	+	0	+	+	+
3	61 F.	Sudden	+	?	+	0	+	+	+
4	75 F.	Sudden	0	?	+	0	+	+	+
5	43 F.	48 hours	+	+	+	+	+	0	+
6	56 M.	Sudden	+	+	+	+	+	+	+

* Time from onset to full development of symptoms.

The symptoms, as shown in Table I, can readily be explained by the neurological lesion, and are accompanied by objective evidence of this involvement as illustrated by Table II. Of the symptoms only vertigo and dysphagia were present in all cases. Pain in the homo-

lateral side of the face, subjective numbness on the contralateral side and dysarthria were present in five of the cases. Diplopia was complained of by four, and cerebellar symptoms were present in three cases. Intractable hiccoughing was present in only two cases.

The neurological signs accompanying these symptoms (see Table II) were as follows: All of the cases showed objective signs of involvement of the spinal fillet, nucleus ambiguus, facial nerve nucleus and the pupillary centre in the medulla. The eighth-nerve nuclei were involved in five of the cases and the cerebellar pathways in three. The descending nucleus and tract of the trigeminal nerve were involved in five of the cases while the main sensory nucleus and motor nucleus of the trigeminal nerve were involved in only one case (Case 6). The abducens nucleus was involved in this case also, and considering the extent of the damage and the finding of blood in the cerebrospinal fluid, it is probable that the lesion was a hæmorrhage rather than a thrombosis.

TABLE II.—Neurological Signs.

Case	Side of lesion	Cranial nerves							Horner's syndrome	Cerebellum	Spinal fillet
		V			VI	VII	VIII	X			
		Motor	Sensory	Descending							
1	R.	0	0	+	0	+	+	+	+	0	+
2	L.	0	0	+	0	+	0	+	+	0	+
3	L.	0	0	+	0	+	+	+	+	+	+
4	R.	0	0	0	0	+	+	+	+	0	+
5	R.	0	0	+	+	+	+	+	+	+	+
6	L.	+	+	+	+	+	+	+	+	+	+

From a study of Table II it is obvious that the lateral portion on the medulla is generally the site of these lesions and the structures located therein, namely, the nucleus ambiguus, spinal fillet, oculo-pupillary nucleus, facial nerve nucleus, and the descending tract and nucleus of the trigeminal nerve, suffer the greatest damage. More dorsolateral structures, as the nuclei of the eighth nerve and the cerebellar pathways (restiform body), are injured with any spread of the lesion.

These cases illustrate several points which are relevant to the anatomical physiology of the hind-brain. In all the cases in which

there was subjective evidence of an involvement of the descending tract of the trigeminal nerve there was a subjective sensation of pain in that side of the face, which in some cases was quite severe. This pain was only transient in our cases, and as yet none have developed any severe trigeminal neuralgia such as that reported by Harris [12]. These cases illustrate the points emphasized by Spiller [14] in 1915, that the descending tract of the trigeminal nerve carries the pain and temperature sense from the face, and that the fibres in the descending tract cross at various levels and join the fibres carrying these types of sensation from the trunk. This last point is brought out in Cases 5 and 6, where the pain and temperature sensation on the contralateral side of the face was lost. The corneal reflex was lost on the side of the lesion in all of our cases, and this is in accord with Spiller's statement that the corneal reflex is connected with the fibres carrying the sensation of pain.

The disparity in the amount of dysarthria and dysphagia found in some of these cases is explained on the basis presented by Ramsbottom and Stopford [13]. They state that the upper portion of the nucleus ambiguus innervates the larynx and the vocal cords, while the lower portion of the nucleus innervates the muscles of the pharynx and the palate. In most of these cases the lesion can be placed fairly low in the medulla, and there was a marked dysphagia with only slight or moderate dysarthria, whereas in Case 6, in which the lesion extended high, there was extreme dysarthria and only slight dysphagia.

The Horner's syndrome is explained by involvement of the pupillary centre, which is located by Combes [3] and others in the lateral reticular substance adjacent to the nucleus ambiguus and the descending tract of the trigeminal nerve. Its connection with the cervical centre is by the tractus intermedio-lateralis. It is also supposed to have a direct connection with the ciliary ganglion by fibres travelling up the descending tract of the trigeminal nerve and out of the brain-stem through the trigeminal nerve. Injury to these cells in the medulla result in a milder form of the Horner's syndrome than that found after a lesion of the cervical sympathetic.

Involvement of the spinal lemniscus was constant in these cases, and was accompanied by a subjective sensation of numbness on the contralateral half of the body; in two of the cases stimulation of the analgesic areas caused a perverted form of sensation somewhat similar to that occurring with injury to higher sensory centres, such as the thalamus.

The sense of taste was tested in only two of these cases, and

in both (Cases 5 and 6) it was found to be absent on the homolateral half of the tongue. This is probably due to injury of the tractus solitarius.

The structures situated in the median portion of the medulla, i.e., twelfth-nerve nucleus, medial lemniscus and the pyramidal tract, are not involved in these cases, as they receive their blood supply through the anterior spinal artery. There was, however, in some of these cases, slight evidence of involvement of the pyramidal tract in the form of hyperactive reflexes, or abnormal plantar or abdominal reflexes on the

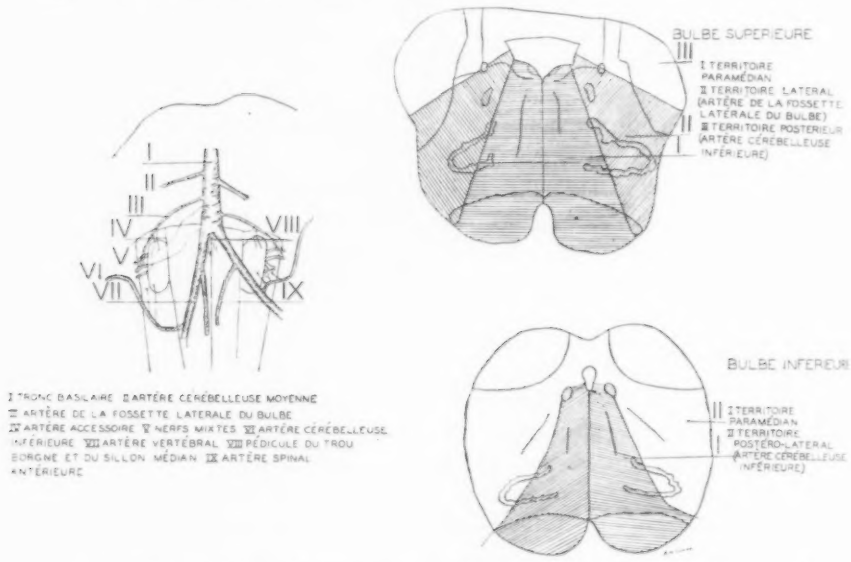

FIG. 9.—Sketches showing the arrangement of the vascular supply to the hind-brain, and the areas of the hind-brain irrigated by each vessel.

side opposite to the lesion. This can probably be explained by œdema, &c. The diminution of deep reflexes which occasionally occurs in these cases (see Cases 4 and 6) has never been explained.

Fig, 9 taken from Foix, Hillemand and Schalit [4], shows their conception of the vascular supply of the hind-brain, and it is their opinion that most of the so-called cases of thrombosis of the posterior inferior cerebellar artery are really due to a lesion of the artery of the lateral fissure.

Diagnosis.—The diagnosis of these cases is generally comparatively simple. The sudden onset of such a dramatic symptom as inability

to swallow should lead to a consideration of a vascular lesion of the medulla, and in the differential diagnosis other causes of injury to the vagus nerve or its nucleus should be considered. The conditions which are most likely to give difficulty are tumours of the medulla, syringobulbia, and the rare cases of osteomyelitis of the temporal bone with involvement of the jugular foramen. The mode of onset and the absence of the concomitant findings of this syndrome, such as the Horner's syndrome, the involvement of the spinal lemniscus and the descending tract of the trigeminal nerve, are generally sufficient to exclude this syndrome. Dysphagia and dysarthria due to the loss of supranuclear control of the nucleus ambiguus, as occurs in pseudobulbar palsy, can easily be distinguished in the same manner.

Prognosis.—The prognosis in these cases, contrary to the current idea that vascular lesions in this situation are fatal, is very good. Though two of our cases died while in the hospital, in only one of the cases could the death be attributed to the vascular lesion, and in the eight cases reported by Riley [9] there were no fatalities. With the progress of time most of the symptoms and signs disappear or improve, with the exception of the sensory changes which are generally persistent.

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HÆMOPHILIA AS A CAUSE OF LESIONS IN THE NERVOUS SYSTEM.

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LESIONS in the central and peripheral nervous system are among the less common local manifestations of hæmophilia, yet they seem to occur often enough to merit consideration in neurological diagnosis. Occasionally the characteristic history of hæmophilia may not be volunteered by a patient, nor elicited by the clinician; and, as in the diagnosis and treatment of joint lesions, a serious mistake may be made through failure to bear in mind this possibility.

The classical memoir of Bulloch and Fildes [1] furnishes us with the most complete historical account of the disease. It contains the records of some nineteen cases in which lesions in the nervous system occurred spontaneously in hæmophiliacs; and it is certain that, in many of the cases, the disease itself was responsible. A summary of these cases follows:—

Mauthner, Max., 1863.—Epistaxis and apoplexy in a man aged 26.
Stromeyer, Louis, 1864.—Paralysis of great sciatic nerve.
Tamele, J., 1869.—Cerebral hæmorrhage.
Traneus, L., 1870.—Ventricular hæmorrhage in a child.
Grandidier, 1872.—Subarachnoid hæmorrhage (Schulz's case).
Lemoine, 1874.—Cerebral hæmorrhage.
de Rute, F., 1884.—Cerebral hemorrhage.
Sarra, R., 1890.—Subarachnoid hæmorrhage in a baby.
Berggrün, Emil, 1896.—Cerebral hæmorrhage.
Pearson, H. B. A., 1904.—Cerebral hæmorrhage.

Family Histories:—

Gocht's case, IV 4.—Subdural hæmorrhage.
Ledoux's case, IV 2.—Hæmatomyelia.
Wright's case 2, III 24, "Edwin."—Cerebral hæmorrhage.
Miles' case, III 2.—Hæmorrhage in the cauda equina.
Bjørkman and Liedbeck's case, II 2.—Hæmorrhage round the pons Varolii, the optic nerves, and the right cerebellar hemisphere.
Van der Scheer's case, 2.—Massive fatal hæmorrhage into the spinal canal.
Forster's case, 2, IV 5.—Meningeal hæmorrhage.
Cousins' case, II 2.—Blood extravasation at the base of the brain.
Getting's case, VI 50.—Lower limb "partially paralysed."

Since 1911, only one case of damage to a nerve due to hæmophilia has been described. J. B. Lord, of Omaha, has given an excellent account of a spontaneous hæmorrhage occurring in a hæmophilic boy, causing ulnar nerve palsy and ischæmic contracture of the muscles of the forearm [2]. A most comprehensive German work on hæmophilia that appeared recently contains no references to lesions in the nervous system [3].

In an attempt to estimate the relative frequency of such lesions, I have examined the records of two large hospitals. In one (St. Bartholomew's Hospital, London) nineteen cases of hæmophilia were admitted to the wards over a period of twenty-three years; in the other (University Hospital, Ann Arbor, Michigan, U.S.A.) ten cases were admitted over a period of fourteen years. The various lesions occurring in these twenty-nine cases are listed below:

	Number	Percentage
Total number of cases	29	100
Epistaxis, bleeding from teeth, minor injuries ..	29	100
Hæmarthroses (generally multiple and recurrent) ..	24	82·8
Hæmatemesis and melæna	9	31·0
Hæmaturia	8	27·6
Hæmorrhage into the eye	1	3·6
Hæmorrhages causing nervous lesions	2	7·2

This table brings out two interesting points: first, that of the major lesions, hæmarthrosis is by far the most common; and, second, that lesions in the nervous system are rare events in the life of the hæmophiliac.

Case Records.

Two cases are described; the first was studied in the Orthopædic Department of the University Hospital, Ann Arbor, Michigan, U.S.A.; the second at the Royal National Orthopædic Hospital, London. The second patient was presented at a clinical meeting of the Neurological Section of the Royal Society of Medicine in November, 1929 [4].

Case 1.—R. H., male, aged 22, labourer.

History.—September, 1928. The patient first complained of soreness followed by numbness in the right groin and the anterior surface of the thigh. The right lower extremity became weak, and some difficulty in walking was experienced. Ten days after the onset of symptoms, a swelling appeared in the right lower abdomen; it was almost painless, but gradually increased in size.

Family history.—Apparently the patient's brother is the only other member of the family with any tendency to bleed spontaneously. No details are given from which one might make a positive diagnosis of hæmophilia in the brother.

Past history.—When aged 10, he had great trouble with epistaxis and bleeding after the extraction of teeth.

When aged 17 his left lower extremity swelled and was painful for two weeks. Later, it was noticed that the limb was shorter than the right, and the patient believes that the hip-joint was a little stiff. The lesion was regarded as tuberculous by his doctor, who ordered him to bed for four months. After this treatment the patient got about quite comfortably, though at first he found it necessary to use crutches.

On examination.—Joints: Left hip, no fixed deformity, active flexion possible to 100°, all other movements very limited.

Right hip, 35° limitation of extension due to contraction of the psoas.

Spine: slight increase in the lumbar lordosis, secondary to the psoas contraction.

Nervous system (all physical signs on the right side).—Sensory: anæsthesia over the areas of distribution of the middle and internal cutaneous nerves of the thigh and the long saphenous nerve.

Motor: no contraction elicited voluntarily or by electrical stimulation in the rectus femoris, vastus externus, vastus internus, crureus, pectineus or sartorius. Complete inability to extend the knee voluntarily.

Abdomen: in the right lower quadrant, lying in the hollow of the ilium, was a mass extending almost to the midline and causing a little bulging in the loin. It was elastic in consistency, had a smooth surface and was not tender.

X-ray examination of the spine was negative. Films of the hip-joints showed considerable destruction of the head of the left femur, and some bony proliferation along the roof of the acetabulum and the margin of the femoral head.

Coagulation time of the blood was ten minutes.

Subsequent history.—By July, 1929, the anterior muscles of the thigh were contracting well; all numbness had disappeared. The patient still walked with a slight limp. The abdominal swelling had disappeared.

On the findings enumerated above, a diagnosis was made of hæmophilia, complicated by hæmorrhage involving the left hip-joint and a right ilio-psoas hæmatoma pressing on the femoral nerve.

There must be an element of doubt in the first part of the diagnosis. The absence of a characteristic family history and the relatively short clotting time of the blood do not suggest classical hæmophilia.

Case 2.— S. L., male, aged 24, chauffeur.

History.—June 9, 1928. The patient was involved in a motor car accident and was admitted to hospital. He was unconscious for three days as the result of "concussion." A radiogram of the spine was said to show a fracture in the region of the third and fourth lumbar vertebræ; there was some injury to the right ankle, but apparently no bony lesion. He remained on his back for four months, and returned to work six months after the accident, not wearing any form of spinal support. At this time the patient noticed that he

occasionally lost control of his legs and fell. There was no actual paralysis and no loss of consciousness.

Early in 1929, he stumbled and injured his right hip. A large swelling appeared in the upper and outer part of the thigh; X-ray examination did not show any fracture of the femur. The swelling gradually disappeared.

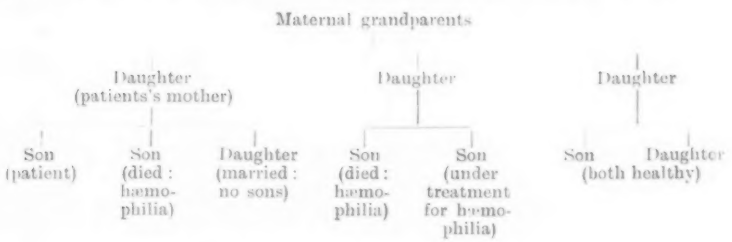
In May, 1929, his right ankle gave way, and he was admitted to hospital with a large swelling of the joint, but again no fracture.

In June, 1929, after a drive of 120 miles (without accident), the patient became aware of a sudden weakness in the left lower limb. There was an almost instantaneous paralysis of the muscles extending the left knee. He experienced a severe boring pain in the left loin, radiating down the left lower limb. This persisted for ten days.

September, 1929, "pins and needles" were sometimes felt in the right lower limb but no muscular weakness.

Micturition was normal and the bowels were kept regular by medicine.

Family history.—The history of hæmophilia is summarized below:—



Past history.—In 1926 the patient was admitted to hospital on account of severe hæmaturia and remained there for six months. There were no marked constitutional signs.

On examination.—Nervous system. Sensory: An area in the left lower limb corresponding fairly closely to the distribution of the anterior divisions of the third and fourth lumbar nerves was insensitive to cotton-wool, pin-prick and differences of temperature. Vibrations of tuning fork were not felt on the left patella, and only felt poorly at the upper and lower ends of the fibula. General diminution in tactile sensibility in the whole of the left lower limb. Joint sensibility everywhere normal.

Reflexes: Biceps, triceps, supinator and ankle-jerks strong and equal on both sides. Right knee-jerk easily obtained, left absent. Abdominal and cremasteric reflexes normal. Plantar reflexes flexor.

Motor: No contraction elicited in left quadriceps extensor muscle, pectineus and adductor magnus. The adductor longus and tensor fasciæ femoris contracted strongly. There was slight wasting of the gluteus maximus.

Recent skiagrams showed no abnormality of the spine.

Lumbar puncture was not performed on account of the blood condition.

The urine was normal.

Clotting time of the blood well over one hour and forty minutes.

When last seen (in December 1929) there was no appreciable alteration in the patient's condition.

Only those muscles supplied exclusively by fibres from the third and fourth lumbar segments were completely paralysed; the sensory loss corresponded to the cutaneous areas for the same segments. It seemed reasonable, therefore, to make a diagnosis of hæmatomyelia at the level of the third and fourth lumbar segments. Probably the injury to the spine in June, 1928, prepared the way for the hæmorrhage.

SUMMARY.

(1) Hæmophilia is occasionally responsible for lesions in the nervous system.

(2) Central lesions are more common than peripheral.

(3) The condition is worth bearing in mind in neurological diagnosis.

(4) Two cases are recorded.

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THE INFLUENCE OF EXPERIMENTAL LESIONS OF THE SPINAL CORD UPON THE KNEE-JERK. I.—ACUTE LESIONS.[1]

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(*From the Laboratory of Physiology, Oxford.*)

[The expenses of this research were defrayed in part by a grant from the Medical Research Council.]

WHEN Ballif, Fulton and Liddell [1] communicated their paper on the differences between "spinal" and "decerebrate" knee-jerks to the Royal Society, the late Sir David Ferrier asked what descending tracts in the spinal cord were responsible for the differences which had been described. The question could not then be answered, and as it has since remained unanswered we have undertaken to investigate the problem and venture now to record our findings. Ballif and his co-workers [1] observed that the knee-jerk of a decerebrate preparation is briefer and always much more difficult to inhibit by a single break-shock stimulus than the jerk of the same preparation after transection of the spinal cord in the dorsal region. This indicates that the "higher centres" exert an influence which tends to abbreviate the jerk and to protect it from inhibition. Denny-Brown [8, 7] has shown that in a muscle composed of mixed fibres, as is the knee extensor, the briefer duration of the "decerebrate" jerk is due to the more ready participation of rapid muscle fibres ("white"). The rapid "white" fibres are inactivated by the spinal severance and thereafter the slower "red"[2] fibres become prominently active. The descending nerve paths responsible for these differences cannot be any of those arising above the level of the colliculi, for the brain-stem had been transected at the mid-collicular level. If transected just posterior to the posterior colliculus so as completely to exclude the red nucleus the result is still the same. Consequently the descending fibres in which we are interested are not

[1] Part II reporting fibre degenerations and myographic findings three weeks after surgical lesions of the spinal cord will be published later. A preliminary note has appeared in the *Journal of Physiology* (1930, **69**, Proc., xxvii-xxviii).

[2] We have used inverted commas since Dr. Denny-Brown [8] was careful to point out that while red fibres are often slow, "redness" in itself does not necessarily imply slowness of contraction.

those in the rubrospinal tract, and they must therefore arise in some centre caudal to the posterior colliculus.

The history of the study of experimental lesions of the spinal cord has been described by Sherrington [31] with special reference to the results of lateral semisection, and by Page May [22] who dealt with experimental lesions in relation to the sensory pathways in the cord. Both authors give full bibliographies of early literature. It will be remembered that Brown-Séquard [2, 3], from an experimental study of semisections carried out between 1847 and 1855, was the first to insist upon the early crossing of fibres conducting pain after their entry into the cord, and upon the uncrossed situation of motor weakness following such lesions. Some of his views were erroneously opposed by Schiff [27] who, however, was among the first to study the consequences of lesions restricted to individual pathways such as the posterior columns. An elaborate series of semisections was reported from Goltz's laboratory in 1882, by Osawa [24], and he it was who described the remarkable fact that two semisections on opposite sides of the cord, if separated by a space of six segments from one another, are followed, after a long interval, by sufficient return of voluntary power to enable the animal to use its hind limbs (somewhat falteringly) for the purpose of progression. In one experiment three semisections were successfully carried out and although all sensation was permanently abolished the animal was eventually able to stand. A series of beautifully circumscribed semisections (monkeys) were reported in 1892 by Mott [23] who drew the important conclusion that whereas pain was diminished below the level of semisection on the opposite side of the body, position sense and touch were lost on the same side. Though certain of Mott's further interpretations have not been substantiated, his conclusions concerning homolaterality of position sense have been amply confirmed. A further series of semisections in monkeys was published by Turner [38]. Gotch and Horsley [16] in the same year studied the effects of acute lesions of the spinal cord upon various reflexes, but the knee-jerk was not analysed. The later studies of Lewandowsky [18], Rothmann [26], Page May [22], and others, in which various quadrants and sensory areas were cut with a view to studying sensory pathways, have proved to have little immediate bearing upon the problem which we now propose to consider. Their work on sensation is fully treated by Head [17], and we do not intend in this paper to deal with literature of the sensory pathways or to consider, except incidentally, the sensory disturbances which our operated animals showed.

Method.

A series of nineteen cats was used in the investigation recorded in Part I of this paper. All animals were decerebrated by the trephine method under profound ether-chloroform anæsthesia. In some preparations (used in conjunction with the study of another problem) the level of transection was well precollicular. When acute lesions were to be made the spinal cord was exposed at a suitable level under anæsthesia, prior to decerebration. After decerebration spinal lesions were made without anæsthesia. The hind-limbs were completely immobilized on both sides by appropriate nerve section or tendon resections, excluding only the nerves to the two quadriceps muscles under examination. Both patellar tendons were isolated, and the preparation securely fixated on both sides to the experimental table so that first one and then the other tendon could be examined by a slight shift of the table. During the first part of the research a torsion-wire myograph of the old pattern was used [33, 34, 15], but the larger number of observations have been made with a new frictionless myograph [5, 12]. It consists of a flat steel strip tightly clamped at both ends and under tension longitudinally. The lever arm is mounted in the middle of the strip, and its excursion is rectilinear, without distortion up to a tension of 6 kg. Its natural vibration frequency is 960 d.v. per sec. Electrical responses have been obtained simultaneously with the mechanical responses [15, 6, 7 &c.] by means of an Einthoven galvanometer. The tendon was tapped two to three times a second by an insulated metal rod held in the hand. Inhibitory stimuli consisted of single break-shocks from a coreless Berne coil, which throughout the whole series of experiments remained at 3 cm., i.e., a strong stimulus, well over maximal for every preparation. When applied to a motor nerve, however, there was no evidence that a stimulus of this intensity ever produced more than one volley of nerve impulses. The peroneal nerve has been used in most instances, and the electrodes were of the Sherrington shielded type. All records were photographic, the technique employed being that previously described in other investigations published from this laboratory [33, 34, 1, 5, 15, &c.]. The spinal cord was removed at the end of the experiment fixed for a week or more in 5 per cent. formalin, and the extent of the lesion examined in thin macroscopic sections.

I.—*Lateral Semisection of the Spinal Cord (low dorsal).*

If a single strong break-induction shock is applied to a nerve of the hind-limb from which a series of knee-jerks is being elicited, e.g., at

3 per second, one jerk may be completely inhibited, provided that the inhibitory stimulus occurs at a brief interval before the jerk (fig. 1). In the decerebrate condition the extensor centres recover within 20 to 30 σ from the inhibition so induced, but when the preparation is made "spinal" as much as 3 seconds may be required before complete recovery [1]. We have therefore used this feature of the difference between the spinal and decerebrate state for the analysis of the pathways. By taking successive observations with intervals at random,

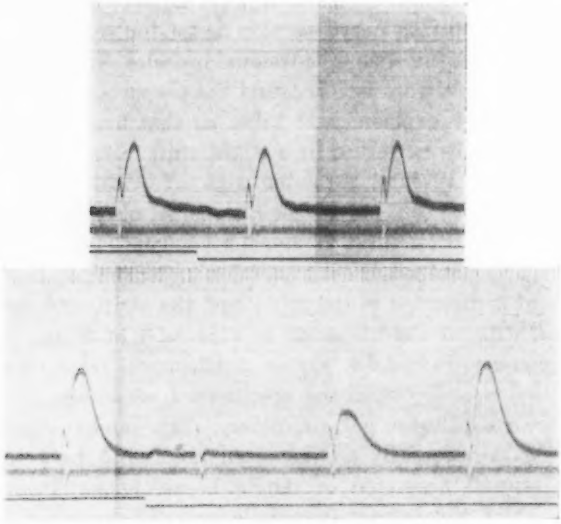


FIG. 1.—Simultaneous electrical and mechanical records of knee-jerks. The taps are seen in the myogram as initial upstrokes preceding the response. An inhibitory break-shock is applied to the peroneal nerve at the fall of the signal at bottom of record. Galvanometer string is between myograph and signal. Time at top of record = 0·02 sec. Upper record: Vasto-crureus in decerebrate cat. Lower record: Same muscle after making a small lesion in ventral quadrant of spinal cord on the side of the muscle in lower thoracic region. This lesion produces a characteristic "spinal" state.

curves can be drawn representing the rate of recovery of the spinal centre from the stimulus (fig. 2). Analysis therefore has consisted in plotting the curves of recovery after any given lesion. We have also studied the duration of the jerk, but as this feature is somewhat more difficult to analyse and to represent graphically it has not been employed. In general, however, it may be said that when the knee-jerk is highly susceptible to inhibition only the more slowly contracting fibres ("red") in the muscle appear to respond [7, 8].

In order to study the effects of incomplete lesions of the spinal cord

it is essential first to analyse the results of complete semisection. If the knee-jerks are examined after an acute semisection, the jerk on the divided side is indistinguishable from the jerk of a preparation in which total transection has been made, while on the uninjured side the jerk remains typically "decerebrate" in all its features. Thus, in Experiment XXI, a precollicular preparation, the knee-jerk was of brief duration and could not be inhibited unless the break-shock fell within 20 σ of the tap. The spinal cord was semisected on the right side, and the jerks on the left still remained "decerebrate" in character, indicating that the descending tracts on the opposite side do not exert an

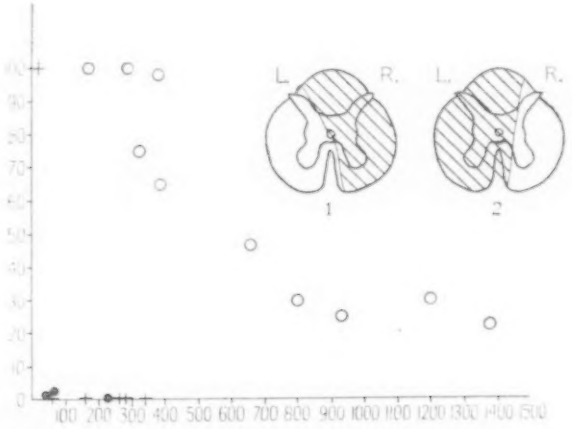


FIG. 2.—Left vasto-crureus. Recovery curve of the knee-jerk after a single inhibitory break-shock to peroneal nerve. Ordinates = degree of inhibitory depression of response expressed as percentage of a normal response to a series of equal taps. Abscissæ = time in sigmata after inhibition. Extent of partial spinal lesion shown diagrammatically. + Before any lesion. ● After right "semisection." ○ After left "semisection."

appreciable influence. (Same result obtained in Experiment VII.) An ipsilateral "semisection" was then performed, after which the jerks became typically "spinal" in character. The curves of recovery before and after these two procedures are indicated in fig. 2, together with diagrams showing the extent of the lesion. It will be seen that after the "semisection" a single shock to a nerve on the same side served completely to inhibit the jerk on that side for 300σ, and a state of partial inhibition continued for 2 seconds. A subsequent complete transection in this experiment caused no increase in the susceptibility to inhibition. We gained similar results in five other preparations.

In some experiments both quadriceps muscles were prepared. On semisection at D.13 the knee-jerk on the same side became immediately "spinal" in character, while that on the opposite side remained "decerebrate." They became indistinguishable from one another after complete transection. In three instances complete transection at mid-dorsal level has been followed by moderate increase in the susceptibility to inhibition on the previously divided side, which suggests that the integrity of the spinal cord on the opposite side may serve to diminish slightly the susceptibility to inhibition (this has also been seen in some experiments in which the cord has been cut aseptically some days

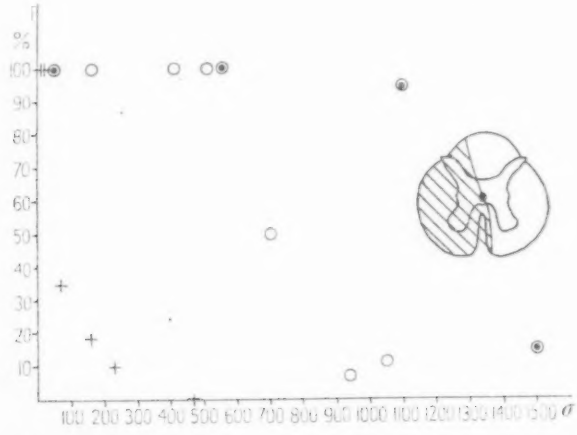


FIG. 3.—Left vasto-crureus. Ordinates and abscissæ as in fig. 2. Extent of left "semi-section" shown diagrammatically. + Before spinal lesion. ○ After left "semisection." ⊙ After complete transection.

before). In Experiment XIX, for example, three distinct curves of recovery from inhibition are seen: (*a*) with the spinal cord intact; (*b*) after ipsilateral semisection at a low dorsal level; (*c*) after complete transection at a low dorsal level (fig. 3). In only one instance has a contralateral semisection noticeably influenced the jerk; in this case, however, the influence was slight, and may possibly have been due to local œdema set up by the operation (Experiment XII).

II.—*Partial Lesions (other than Semisections).*

Since a semisection of the spinal cord serves to make the knee jerk on the same side "spinal" in character, we were led to consider the

results of incomplete semisections. Those performed acutely were found of significance only when the inhibitability of the knee-jerk remained unchanged. When it became "spinal" in character the result might be due to local œdema or to direct trauma affecting the unsevered parts of the cord. Difficulties of interpretation arising from this source are not present in chronic lesions, for the œdema usually passes off, and the extent of the primary lesion can be judged by the resulting degenerations. There are, however, other complicating factors in the chronic preparations, especially the inevitable compensation which occurs [31, 35, 36]. We are therefore inclined to place significance upon the results of acute lesions.

In each of a series of ten animals, one or more partial transections have been made, and the effect of each lesion on the knee-jerks has been carefully analysed. We propose to enumerate these with diagrams in the order in which they were performed.

Experiment II.—Precollicular preparation; right quadriceps recording, giving at first normal "decerebrate" jerks, difficult to inhibit. After a lesion which subsequently was found to involve the dorsal two-thirds of the spinal cord on both sides at D.10, there was no change in the duration of the jerk (90σ), and the inhibitability was only slightly increased. The extent of the lesion is shown diagrammatically in fig. 4. After complete transection of the cord the jerks increased in magnitude (4 mm. to 19 mm.) and duration (90 to 160σ) and the inhibitability of the jerk was increased more than twofold.

FIG. 4.—Description in text.

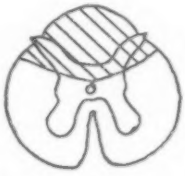
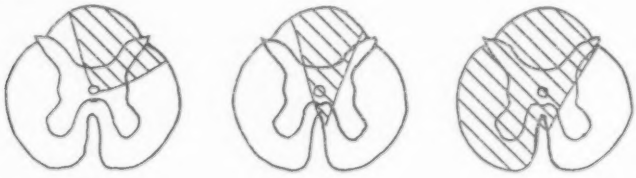


FIG. 5.—Description in text.

Experiment III.—Precollicular preparation; right quadriceps recording with normal "decerebrate" jerks. A lesion at D.9 affecting the posterior columns on both sides, the dorsal part of both lateral columns and extending downward as far as the central canal (fig. 5) had no effect on the inhibition or on the duration of the jerk. Dial was given to this preparation intraperitoneally (0·5 c.c. per kilo) and it served only to make the knee-jerk more difficult to inhibit. The spinal cord was not transected.

Experiment IV.—A precollicular preparation; right quadriceps recording giving characteristic, brief "decerebrate" jerks difficult to inhibit. At intervals of 30 to 40 minutes three lesions were made at levels ranging from D.7 to D.10

(see fig. 6), the first in the upper third of the right dorsal quadrant; the second was a stab wound affecting a triangular area involving the grey matter and posterior columns; and the third an extensive lesion involving the whole left side of the cord and the right dorsal quadrant. These three lesions were each without any recognizable effect upon the duration (60σ) or the inhibitability of the jerk. Ten minutes after complete transection the duration of the jerk had risen to 180σ and the recovery curve from inhibition had become characteristically prolonged.



1 2 3

FIG. 6.—Description in text.

It is quite clear then from these three preliminary experiments that pathways controlling the extensor centres pass downwards in the ventral quadrant of the spinal cord and do not cross below the level of our lesions. Subsequent efforts to localize these tracts more accurately have been suggestive but not conclusive. Acute experiments have been somewhat variable, possibly because of trauma incident to the transection and probably also because the descending tracts are scattered diffusely through the ventral quadrant of the cord. The next experiment suggests that the descending tracts are perhaps not sharply circumscribed.

Experiment V.—Midcollicular preparation with good decerebrate rigidity; right quadriceps recording and yielding brief "decerebrate" jerks difficult to inhibit. An extensive lesion (fig. 7, No. 1) involving the dorsal two-thirds of the right half of the spinal cord was followed by considerable increase in susceptibility to inhibition. Subsequent complete semisection (fig. 7, No. 2) had little further effect, but complete transection increased susceptibility to inhibition on the right side by 20 per cent. to 30 per cent. It is likely that this experiment had been complicated by local œdema.

Having found that the dorsal quadrant of the spinal cord could be transected rostral to the extensor centres of the hind-limb without influencing the jerk, we next attempted to produce localized lesions of the ventral quadrant of the cord in order to learn whether they would influence the jerk when the rest of the cord remained intact. Lesions of the ventral quadrant are difficult to place accurately since the approach to the cord is from the dorsal surface. In successful instances it has appeared evident that circumscribed destruction of this area of the

spinal cord does in fact cause a "decerebrate" jerk to become "spinal" (fig. 1). For the reason mentioned at the beginning of this section positive evidence drawn from acute lesions is, however, less satisfactory than negative evidence. Two representative experiments will be described, in the first of which the evidence is positive, in the second significantly negative.

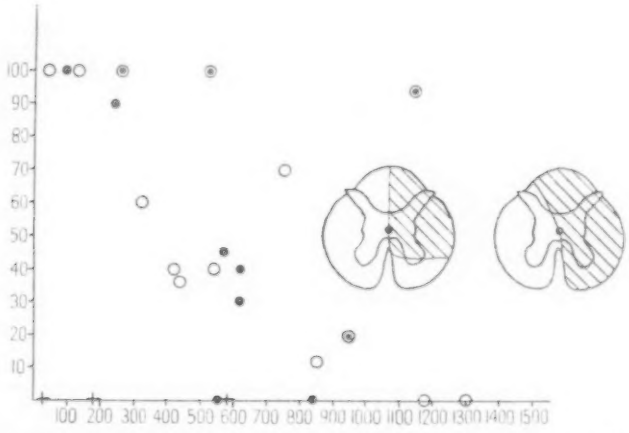


FIG. 7.—Right vasto-crureus. Ordinates and abscissæ as in fig. 2. + Before spinal lesion. ● After section of right dorsal quadrant lesion (1). ○ After semisection, lesion (2). ⊙ After complete transection.

Experiment XIII.—Precollicular preparation; left quadriceps recording. Before any lesions of the cord were made the curve representing the recovery from inhibition of the jerk was characteristic of that of a precollicular preparation. After an extensive lesion restricted to the right ventral quadrant of the spinal cord at D.9, there was no increase in the susceptibility to inhibition of the jerk, the curve being that usually seen in a typical decerebrate preparation (fig. 8). After a second lesion, this time restricted to the left ventral quadrant (at D.10), the knee-jerks on the left side became typically "spinal" in character, becoming enhanced in duration from 60σ to 90σ, and showing a recovery cure in which the size of the jerk was still 50 per cent. below normal one second after a single inhibitory stimulus. On subsequent complete transection of the cord at D.8 the recovery curve of inhibition and the duration of the jerk were not changed significantly.

This experiment indicates that a lesion confined to the ventral quadrant on the same side as the jerk serves to make it "spinal" in character.

The next experiment was similar, only it so happened that the second lesion was incomplete, failing to extend to the midline. The

conclusion to be drawn was that the nerve-fibres with which we were concerned passed downwards in a medial sector of the ventral quadrant.

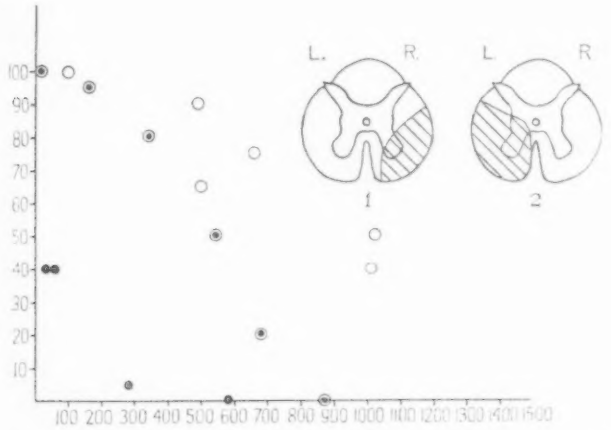


FIG. 8.—Left vasto-crureus. ● After right ventral quadrant lesion (1). ○ After left ventral quadrant lesion (2). ⊙ After complete transection.

Experiment XVIII.—Precollicular preparation: left quadriceps recording. Before a lesion of the cord had been made, recovery from a single shock was complete after 50 to 100σ. This was also true after a large lesion of the right ventral quadrant extending to the midline. After a lesion of the left ventral

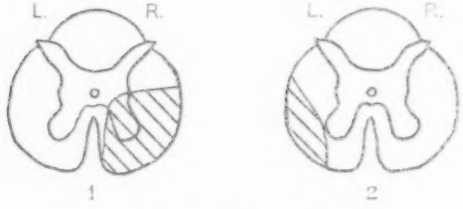


FIG. 9.—Description in text.

quadrant which extended to within about 1 mm. of the midline there was still no change in the recovery curve from inhibition. After the cord had been completely transected, however, the characteristic "spinal" curve resulted and the duration of the jerk became increased from 60 to 140σ.

Acute experiments then point definitely in one direction; namely, that the fibres responsible for the "decerebrate" characteristics of the jerk pass downwards in a medial sector of the ventral quadrant in an

area which would include the vestibulospinal pathway. When this region is cut not only does the jerk become spinal in character but the crossed extensor reflex disappears. Further observations, derived from the study of "chronic" preparations, will be reported later.

III.—*The Influence on the Knee-jerk and Stretch Reflex of Transection of the Cord caudal to the Segments supplying M. quadriceps.*

An observation long familiar to those who have worked with decerebrate preparations is that on transection of the spinal cord in the dorsal region the decerebrate rigidity of the fore-limbs becomes very greatly augmented. The effect is a lasting one, being seen a few minutes after transection and continuing some days after decerebration. It would appear that the centres of the fore-limbs are inhibited normally by impulses ascending from the hind-limbs, and that transection of the cord serves to release them. In analysing this effect more in detail, Denny-Brown and Liddell [9, pp. 362-363] found that the stretch reflex of M. Supraspinatus is greatly increased by post-brachial severance of the spinal cord, and they draw attention to Sherrington's and Lazlett's [36] observations that the direct "spino-cerebellar" tract becomes diminished in size as it ascends the spinal cord. Undoubtedly there are also other tracts which transmit influence from the more caudal segments. Fröhlich and Sherrington [14], for example, found that stimulation of the transverse cut surface of the spinal cord in the lumbar region with stigmatic electrodes caused inhibition of decerebrate rigidity in the fore-limbs when a circumscribed area just lateral to the ventral horn was stimulated. Changes in position of the hind-limbs produce alterations in the posture of the fore-limbs [25].

These observations suggested that even in the case of the hind-limb the more caudal segments of the cord might exert an inhibitory influence upon the rostral extensor centres: and that, for example, transection of the cord at the sixth or seventh post-thoracic level might diminish the susceptibility to inhibition of the "spinal" jerk. In 1893 Sherrington had observed [19] that "the briskness of the 'jerk' may in like manner be increased by section in the spinal canal of the spinal roots, either sensory or motor, adjacent to that one most intimately concerned with the jerk, and especially of the muscular nerves to the antagonist of the 'jerk's' muscles," but transection of the cord was not examined in these experiments. Our procedure has been to examine first the jerks in an ordinary decerebrate preparation, after which the spinal cord has been transected in the dorsal region and the jerks and their inhibition again

examined. Soon after spinal transection and for about twenty-four hours subsequently, it is usually impossible in the cat to obtain a stretch reflex (this being one of the prominent manifestations of spinal shock). Having found the stretch reflex to be absent, the spinal cord has then been transected at some level caudal to L.5.

In one experiment (XXXVI) in which the cord was transected between L.5 and L.6 all of the nerves from muscles antagonistic to quadriceps being thus excluded, one of the prominent symptoms of spinal shock, i.e., the absence of stretch reflex, disappeared. We were not able in this experiment to test the susceptibility of inhibition since the chief

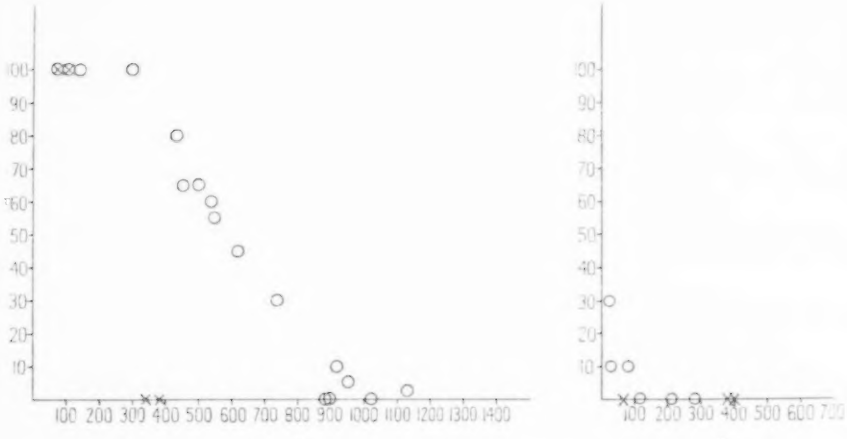


FIG. 10A.—Right vasto-crureus. ○ After right semi-section at L.1.
× After complete transection at L.6.

FIG. 10B.—Same preparation. Left vasto-crureus. ○ After right semisection at L.1.
× After complete transection at L.6.

afferent pathways had been cut. In the following experiment the transection was made between L.6 and L.7 so that one, at least, of the large caudal segments from which the sciatic nerve arises was left intact.

Experiment XXXVII.— Midcollicular preparation with right semisection at L.1 before observations were made. The recovery curve of the right quadriceps was "spinal" in character, while on the left it was typically "decerebrate" (fig. 10, A and B). The spinal cord was then transected between L.6 and L.7, after which there was a very marked increase in the briskness of the jerk on the right side with reappearance of stretch reflex, and the recovery curve had moved to the left of the chart (fig. 10A). Before the lower lumbar transection

recovery on the right had not been complete until 900 to 1000σ, whereas after the transection recovery was complete after 340σ.

Though it is difficult to exclude altogether the possibility that the result is due to a diminution in the total number of effective fibres from the afferent nerve used for stimulation, it is improbable that this would account entirely for the result, since it is well known that a small nerve may be quite as effective in producing inhibition as a large one. The result, therefore, suggests that transection of the spinal cord caudally affects the three major symptoms of spinal shock: (1) susceptibility to inhibition; (2) the stretch reflex; (3) the crossed extensor reflex (see Part II).

Similar observations have been made in four other experiments with minor modifications of procedure. We have found in addition that section of the cauda equina at the tenth post-thoracic level is without influence upon spinal shock. Similarly, section of the left posterior roots L.7 and L.8 had no demonstrable influence upon the knee-jerks of the right quadriceps. Transection of the cord at the eighth post-thoracic level appeared to exert a slight influence. From these observations it appears to us that a transection of the cord which removes the influence of the antagonist tends very markedly to diminish the symptoms of spinal shock in the quadriceps.

Discussion.

Our observations offer two points of interest to the clinical observer: (1) That "spinal shock," so-called, in so far as it affects the knee extensor, is a condition associated with a very marked increase in the susceptibility to inhibition, as judged by the knee-jerk and the crossed extensor reflex; and (2) that this "spinal shock," in the cat at least, does not develop until the descending pathways in the ventral quadrant of the spinal cord have been disturbed. It appears, also, that the more important pathways descend in a medial sector of the ventral quadrant in a region which would include the vestibulospinal tracts. So long as these are intact the limbs retain their extensor posture, and the knee-jerk is brisk, brief and difficult to inhibit. We have a few observations which suggest that the neck and labrynthine reflexes still exert their effect upon the knee-extensor. The observations are in harmony with those made by Cairns and Fulton [4], who found when radon was applied to the spinal cord (in a dose of 5 millicuries or greater) that the animals passed through a stage of marked hyperextension of the hind-limbs, lasting sometimes as long as twenty-four hours,

before passing into a flaccid paraplegia. In one of their animals which remained permanently in extension on one side, the descending pathways in the medial sector of the ventral quadrant on that side had been spared. In such preparations the scratch reflex is still elicitable from stimulation behind the ear, which is in harmony with the observations of Sherrington and Laslett [35]. In his studies of tonus of skeletal muscle Spiegel [37] found that lesions of the ventral cord produced a marked and long-continued hypotonus of the skeletal musculature on the same side, whereas lesions of the lateral and dorsal columns produce only a slight and transient hypotonus of the homolateral musculature.

The fact that the vestibulospinal pathways appear to be responsible for the differences between the "decerebrate" and "spinal" jerk is in harmony with the experiments of Magnus [21, p. 434; see also 15, p. 530], that decerebrate rigidity continues to exist so long as the transection of the brain-stem does not injure the vestibular nuclei, i.e., it must fall somewhere between the anterior colliculus and the level of the vestibular nuclei. Immediately the vestibular nucleus is excluded decerebrate rigidity vanishes. If pathways other than vestibulospinal are concerned in causing the difference between the spinal and the decerebrate condition, they must arise at or caudal to the vestibular area.

Our conclusion is that spinal shock is characterized by the more or less permanent existence within the spinal cord of increased susceptibility to inhibition from external stimuli. Whether this is due to an intrinsic increase of the central inhibitory state of all or certain neurones, or to an intrinsic decrease of their central excitatory state, is a problem which awaits investigation.

Summary.

(1) One of the major physiological symptoms of spinal shock is an increased susceptibility to inhibition. The knee-jerk, for example, may be inhibited for a period of several seconds by applying a single break-induction shock to a sensory nerve of the hind-limb. In the intact animal, or in a decerebrate preparation, it is exceedingly difficult and often impossible to inhibit a knee-jerk by a corresponding stimulus. The present paper records a series of experiments in which the attempt was made to determine the pathways responsible for the differences between the decerebrate and spinal condition, as judged by the inhibition of the knee-jerk.

(2) Acute lesions were made at levels ranging from D.8 to L.1, after which knee-jerks were examined by means of a frictionless torsion-wire myograph of high natural vibration frequency, with simultaneous registration of the electrical response according to the technique described by Ballif, Fulton and Liddell.

(3) Complete lateral semisection of the spinal cord causes the knee-jerk on the side of the semisection to assume "spinal" characteristics, while the jerk on the opposite side remains "decerebrate."

(4) The dorsal two-thirds of the spinal cord may be cut through without causing the knee-jerk to become "spinal" in type on either side.

(5) In acute preparations isolated transections of the ventral quadrant of the spinal cord on one side cause the knee-jerk on the same side to become "spinal."

(6) We conclude from these observations that the descending pathways responsible for the differences between the "decerebrate" and "spinal" conditions pass in the ventral quadrant of the spinal cord, probably in the medial half. The vestibulospinal tracts therefore suggest themselves since they are the most important descending fibres in this area.

(7) Transection of the spinal cord caudal to the knee-jerk centres causes great enhancement in their activity, diminution in susceptibility to inhibition, and, in a "spinal" preparation, the prompt reappearance of the stretch reflex.

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THE INFLUENCE OF UNILATERAL DESTRUCTION OF THE VESTIBULAR NUCLEI UPON POSTURE AND THE KNEE-JERK.[1]

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It has been known for many years that the level of the brain-stem corresponding with the vestibular nuclei is essential for the maintenance of decerebrate rigidity [14, 16, 3, 10, 12], but precise information concerning the source and nature of the influence exerted by this area upon the spinal cord is lacking. In a previous communication [4] we pointed out that decerebrate rigidity continued to exist as long as the ventral quadrant of the spinal cord on the same side remained intact, and that when this area was destroyed reactions below the level of the lesion became spinal in character, even though the opposite lateral half of the spinal cord was uninjured. Ballif, Fulton and Liddell [2] drew attention to three important physiological criteria of the acute spinal condition: (i) Great depression of stretch reflex with disappearance of decerebrate rigidity; (ii) increased inhibitability of the knee-jerk; and (iii) prolongation of the jerk due, as is now known, to predominant participation of "slow" "red" muscle fibres after transection of the cord. Since the vestibulospinal tract is the chief descending pathway in the region of cord found essential for decerebrate rigidity, we have undertaken to destroy the tract at its origin by making lesions in the vestibular area of the medulla, and we propose to describe the results in the present paper.

Method.

Destruction of the vestibular nuclei.—Certain previous writers Gray [7]; Papez [11]; Allen [1], &c., have employed the method of inserting a probe perpendicularly through the cerebellum into the brain-stem, at a point corresponding to Deiters' nucleus. Although this

[1] A preliminary note under the same title has appeared in the *Journal of Physiology* (1930, **70**, *Proc.*, xvii).

operation has the advantage of being relatively simple surgically, it is undesirable since accuracy is difficult, and destruction of cerebellar tissue is entailed as well as the likelihood of injury to deeper structures in the medulla, e.g., rubrospinal tract. In an investigation on the cerebellum [6], we found that a satisfactory approach to the floor of the fourth ventricle could be secured under "dial" anæsthesia [5] by exposing the caudal half of the cerebellum and gently elevating the posterior vermis. With the medulla thus uncovered we found that by means of a small, slightly-curved red-hot needle about 1 mm. in diameter, it was possible to make a satisfactory lesion in any part of either vestibular region. Injury to the cerebellar peduncles as well as to the posterior longitudinal bundle and the gracilis and cuneate nuclei can be avoided by this technique. The details of our surgical approach are fully described in our forthcoming paper on the cerebellum [6].

Recording.—In acute experiments, vestibular lesions have generally been made under profound anæsthesia immediately after decerebration; but in some experiments the lesions were made some hours after the animal was decerebrated. The usual dissections were then made previous to myographic examination, i.e., isolation of both quadriceps muscles and complete denervation and immobilization of all other muscles in both hind limbs. Photographic records of responses were obtained with the new frictionless torsion-wire myograph [4], simultaneous electrical records being made with an Einthoven string galvanometer in the same optical system. For inhibition maximal single break-shock stimuli from a coreless Berne inductorium at 3 cm. applied to the ipsilateral popliteal nerve were used in all experiments. The stimuli were applied by means of Sherrington glass-shielded electrodes.

In our "chronic" animals, daily clinical observations were made before the myographic examination, which took place at the end of three weeks. After myographic examination, the spinal degenerations were studied by Marchi's method with no essential modification, except for prolonged washing after the tissue had been removed from dichromate. As yet only the spinal degenerations have been studied in detail and the medulla and cerebellum have been preserved to await detailed anatomical analysis. Since such factors as local œdema, &c., cannot be controlled in fixed preparations, the extent of acute lesions has only been studied macroscopically in fixed material. In one instance, however, we cut serial sections 20 μ thick through the region of the lesion after formalin fixation, staining every tenth section with hæmatoxylin and eosin.

I.—Acute Experiments.

If, when decerebrate rigidity has appeared after mid-collicular transection of the brain-stem, a small lesion is made by inserting the red-hot needle into the medulla oblongata at a point 2-3 mm. from the midline at the level of the lateral angle of the fourth ventricle, the rigidity on the side of the lesion becomes much less within 1-2 minutes. If the lesion is made within 1 mm. of the midline little or no change is likely to occur. When the lateral area has been destroyed in this way, the resulting asymmetery may be so marked that if the preparation is supine the two limbs opposite to the lesion may be rigidly extended, while those on the same side lie in a flaccid, flexor posture on the surface of the body. To inspection the knee-jerk on the injured side is pendular in character, as in human cases with hypotonia. When examined myographically the quadriceps muscle shows a diminished or completely absent stretch reflex on the injured side, and the jerks tend to be more prolonged in duration as a result of the predominant participation of the red-fibred constituents of the muscle. Occasionally, following a vestibular lesion, we have observed "mixed" jerks in which a distinct white-fibred component is seen followed by a delayed relaxation characteristic of the red-fibred component and quite distinct from the "hump" of decerebrate rigidity, the latter being accompanied by action currents while the red-fibred "delay" shows no corresponding disturbance in the electrical record. Another significant distinction is that on the injured side the knee-jerk is more readily inhibited by single break-shocks, but the susceptibility to inhibition is less marked than in a spinal preparation. For comparison the curve of recovery of the jerk following a single inhibitory shock can be readily obtained on the two sides, as in the following figure taken from the experiment described below. It will be seen that on the intact side a single inhibitory shock is effective for less than 600σ, while on the injured side it is effective for $1{,}000\sigma$.

All our experiments have given essentially the same results, and the ensuing protocol will serve to illustrate the character of the observations.

June 17, 1930.—A young cat, weight 2 kg., was prepared under profound ether-chloroform anæsthesia, the two hind limbs being completely denervated except for the two quadriceps muscles.

Lesion in the right Deiters' area.—The cerebellar region was exposed by separation of the neck muscles, and the floor of the fourth ventricle brought into view by elevation of the posterior vermis. The preparation was then decerebrated at the mid-collicular level and after decerebrate rigidity had come on a

curved red-hot needle was carefully inserted into the medulla at a point 1·5 mm. to the right of the midline, and was carried outward about 2 mm. from this point toward the apex of the lateral angle of the fourth ventricle. Respiration stopped momentarily, but was resumed again without recourse to artificial respiration.

Clinical observations.—Within three to four minutes after the lesion there was an evident diminution in the rigidity on the right side and the difference between the two sides became more and more marked in the course of the next

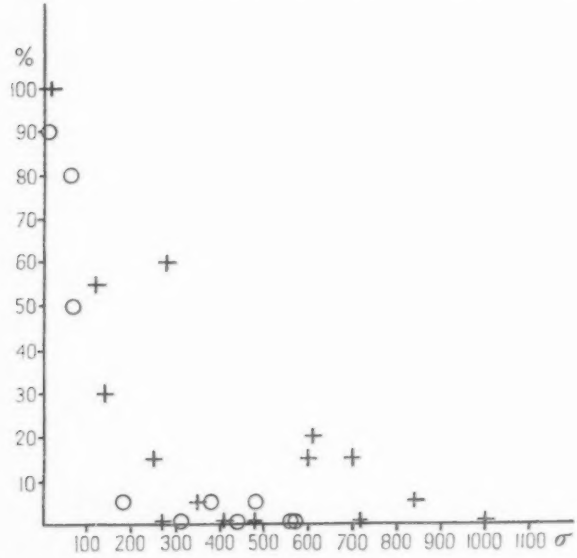

FIG. 1.—Acute experiment, described in text. Injury of medulla oblongata on right side. Ordinates — degree of inhibitory depression of response expressed as percentage of a normal response to a series of equal taps. Abscissæ — time in sigmata after inhibition. + Right vasto-crureus. ○ Left vasto-crureus.

hour. Three hours after the lesion had been made the left fore-limb was rigidly extended, and the right fore-limb lay flaccidly on the table. The difference persisted in all positions of the animal. The general condition of the preparation in respect to heart and respiration was entirely satisfactory and the pinna and conjunctival reflexes were active. The left knee-jerk to inspection was quicker than the right, and the right showed the pendular fling characteristic of hypotonia.

Myographic examination.—When attached to the myograph the stretch reflex of the right quadriceps, though present at first, was not sustained and soon vanished completely. The knee-jerk was large, slow and required a high initial tension for its elicitation. There was no trace of a white-fibred

component. The jerk was readily inhibited and the curve of recovery following a single break shock inhibition is shown in fig. 1. The crossed extensor reflex was difficult to elicit and of brief duration, exhibiting but little after-discharge.

The left quadriceps on the other hand showed quick brisk knee-jerks, a marked and well-sustained stretch reflex and the jerks were considerably more difficult to inhibit than on the right side, but it appeared that they were more susceptible than the jerks of a normal decerebrate preparation. Crossed extensor reflexes were brisk and well-sustained.

Neck and labyrinthine reflexes.—Despite the fact that the neck muscles had been freely resected, turning of the head caused evident effect in the *left* quadriceps, there being a spontaneous increase in rigidity on turning it maximally and diminution when in the minimal position. With the *right* quadriceps the neck reflexes were only just present and were scarcely perceptible, maximal turning being required to produce 50—100 grm. spontaneous increase in tension.

Pathological note.—The medulla oblongata and upper spinal cord were fixed in formalin and embedded in paraffin. Serial sections 20μ thick were cut and every tenth section mounted and stained with eosin-hæmatoxylin. The lesion involved the right dorso-lateral surface of the medulla, extending from the rostral poles of the nuclei of Goll and Burdach forward into the middle peduncle of the cerebellum. The whole of the descending vestibular nucleus and the caudal third of Deiter's nucleus were completely destroyed. More rostrally the dorsal portion of Deiter's nucleus, the nucleus of Bechterew and the lateral portion of the medial vestibular nucleus were destroyed. The lesion cut the restiform body and in the middle cerebellar peduncle extended dorsally to the ventral portion of the nucleus emboliformis. Only the most rostral fibres of the uncinate fasciculus of Russell escaped.

II.—Chronic Experiments.

With aseptic precautions the region of the right vestibular nuclei was cauterized in four cats as described above. The protocols of two of the experiments are given in detail below. All four of the animals showed the usual clinical picture, of which the most prominent features were as follows: nystagmus, forced rolling movements to the side of the lesion, asymmetrical postures, and hypotonia of both homolateral limbs. In all cases the intensity of the symptoms became considerably less marked with time.

Animal II.

Operation, May 2, 1930.—Under "dial" anæsthesia the usual cerebellar exposure was made, and the posterior lobe of the vermis was elevated. The red-hot needle was then thrust gently into medulla at a point 2 mm. to the right of the midline slightly caudal to the lateral angle of the fourth ventricle. The needle was inserted a second time to destroy some of the superficial area

around the first lesion. There was no bleeding and the wound was closed as usual in layers.

Post-operative course.—The animal began to come out of the anæsthetic within twenty-four hours and took fluid and solid food after two days. Its general condition and nutrition remained excellent. There was slight weakness of the right side of the face for three or four days, with complete recovery from this within a week.

Forced rolling movements developed as the anæsthetic wore off and were most marked for the first four days after operation. During this period, when placed on the floor, the cat would roll to the right until its progress was impeded by some obstruction. From the fifth day the rolling movements became gradually less, and had practically stopped by the thirteenth day. The *posture* was asymmetrical throughout the period of observation, the head being turned with the occiput to the right, the chin to the left. For the first few days the cat braced itself against the back of the cage, but later it lay curled up with the head and tail bent to the left, although the head remained rotated as described above. A rapid rotatory *nystagmus* appeared as the anæsthetic wore off, the quick component being clockwise, the slow component anticlockwise. This was very marked and almost continuous for the first four days, but then gradually diminished and disappeared between the eighth and twelfth days. There was marked *hypotonia* of the right fore and hind-limbs on the first day after operation, which continued throughout the period of observation, although it was less marked at the end of two weeks. The *knee-jerks* showed a similar difference, the left being noticeably brisker and the right being pendular. A slow shaking *tremor* of the head and shoulders appeared as the rolling movements decreased and remained present throughout the period of observation, although it was never a very marked symptom.

On the last day of observation, the twenty-first day after operation, the animal was able to walk in a crouching position, the trunk and tail held stiffly, the head rotated slightly (chin to the left). It was very unsteady, frequently falling to the right. There was a definite limp on the right hind leg. When held up by the scruff of the neck this leg hung lower than the left, although the tone of the fore-limbs was approximately equal. The right knee-jerk was pendular, and the scratch reflex on the right showed a longer latent period than that on the left. There were only occasional nystagmoid jerks on lateral deviation.

Myographic examination. May 23.—Under deep chloroform-ether anæsthesia the preliminary dissection of the hind limbs was made after which the cat was decerebrated by intercollicular section through a trephine opening in the skull. Good extensor rigidity developed within ten minutes of decerebration and was definitely more marked on the left than on the right. The general condition of the preparation with regard to heart and respiration remained good throughout the period of observation.

Three hours after decerebation the preparation was arranged for myographic recording. Observations were made first from the right and then from the left

quadriceps. At the end of the experiment it was again noted that on holding the preparation symmetrical and supine the left fore-limb showed good extensor rigidity, while the right fore-limb sunk down partially flexed.

A *stretch reflex* was present on both sides, but definitely more pronounced left than right. Inhibition of the stretch reflex on stimulation of the ipsilateral peroneal nerve was less complete on the left.

The *knee-jerk* on the right was of long duration, and there was rather marked inhibition following stimulation of the ispilateral nerve. On the left the jerks were of shorter duration and were somewhat irregular, but the

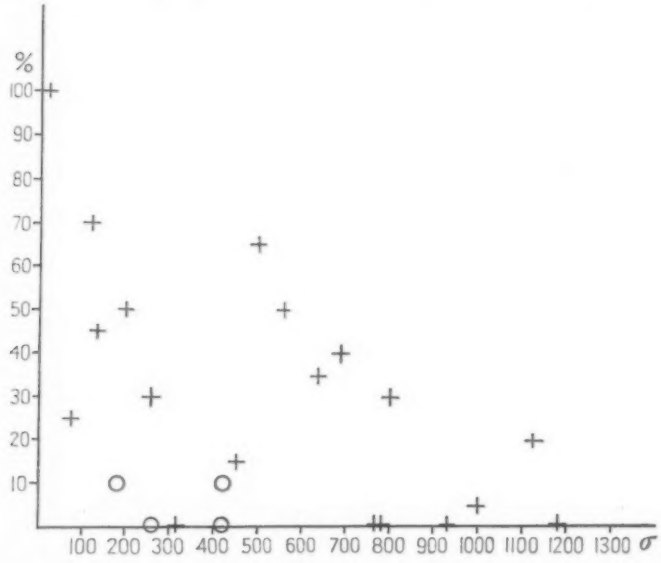

FIG. 2.—Chronic experiment, Animal II, described in text. Injury of medulla oblongata on right side. Ordinates and abscissæ as in fig. 1. + Right vasto-crureus. o Left vasto-crureus.

inhibition was less pronounced. A comparison of the rate of recovery from inhibition may be made by reference to fig. 2.

The *crossed extensor reflex* was observed on both sides, the response of left muscle being smaller than that of the right. With the head in the intermediate position the response was small and quick, but with the head rotated either toward or away from the muscle, the response was large and longer.

The *labyrinthine and neck reflexes* were repeatedly observed in both muscles and it was constantly found that with the head turned to the maximal position the resting tension in the muscles was higher, and there was less inhibition of the jerk, but with the head in the minimal position, the resting tension of the muscles was lower and the jerk somewhat more readily inhibited.

Pathological note.—Macroscopical examination of the medulla showed a scar in the right dorso-lateral region which involved the vestibular nuclei, the restiform body, and the middle portion of the middle peduncle of the cerebellum. The brachium conjunctivum was intact. Sections from the cervical, thoracic and lumbar cord, stained by the Marchi method, showed heavy degeneration in the direct vestibulospinal tract on the right side (fig. 3.) There were less heavy

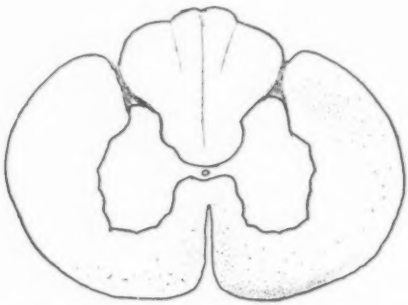

FIG. 3.—Degenerations in Animal II from the cervical level.

degenerations in the marginal fasciculus, in the region of the medial reticulospinal tract of Papez [11], and of the descending vestibular component to the medial longitudinal fasciculus of the homolateral side described by Rasmussen [13]. In the cervical section there were also scattered degenerations in the region of the crossed vestibulospinal tracts of Gray [7], and the region of the homolateral rubrospinal tracts, but these were no longer seen in the sections from the thoracic cord.

Animal V.

Operation. May 14, 1930.—A cerebellar exposure was made under "dial" anæsthesia and the posterior lobe of the vermis elevated. The right vestibular area was then cauterized by twice inserting the red-hot needle about 2 mm. from the midline, approximately at the level of origin of the eighth cranial nerve. There was no change in respiration and no bleeding. The incision was closed in the usual manner with discontinuous silk sutures.

Post-operative course.—The animal began to come out of the anæsthetic in thirty-six hours, and took food and fluid on the second day. Its general condition remained excellent throughout the period of observation. *Forced rolling movements* appeared as the anæsthetic wore off, and were very marked from the third to the fifth day after operation, the animal rolling to the right across the floor until it was stopped by some obstacle. Compensation occurred rapidly, and by the eighth day the animal was able to sit in a horizontal position, although there was some swaying, and it was apparently dizzy. The *posture* during the period of observation was asymmetrical, the head being rotated, and also bent to the right, but there was no tremor.

For the first six days post-operative there was a spontaneous lateral *nystagmus*, the quick component to the left, the slow component to the right. This symptom then subsided. *Hypotonia* was present throughout the post-operative period in both limbs of the right side, being more marked in the hind-limb than in the fore-limb. Comparing the two hind-limbs, the *knee-jerks* remained equally brisk, but on the right they were pendular, whereas on the left they were normal. The *righting reflexes* were normal after the first ten days. If the cat was placed on its back it came to the horizontal position by turning either to the right or to the left, whichever direction was shorter.

On the last day of observation—the twenty-third day after operation—the animal was able to run in a half-crouching position, the body at an angle of 15-20° with the direction of the line of motion. It was unsteady and frequently fell, usually to the right, but occasionally to the left. There was definite limp of the right hind leg, and several times this leg collapsed under the weight of the body. When held up by the scruff of the neck there was a slight tendency to bend towards the right. The knee-jerks were active and essentially equal, there was no positive supporting reaction, and the scratch reflex was slightly more active on the left than on the right.

Myographic examination. June 6, 1930.—Under deep chloroform-ether anæsthesia the preliminary dissection of the hind-limbs was carried out, after which the cat was decerebrated. Good decerebrate rigidity developed in fifteen minutes, and was much more marked on the left side than on the right. Three hours later the preparation was attached to the myograph for recording first from the right and then from the left quadriceps in the same manner as in previous experiments. The general condition of the preparation remained good throughout the period of observation, and the difference in rigidity of the two fore-limbs was unaltered.

There was a slight *stretch reflex* present in the left quadriceps which was only very slightly inhibited by stimulation of the ipsilateral peroneal nerve. On the right the stretch reflex was slightly larger, but it was markedly inhibited by the same strength of stimulus. The *knee-jerks* were very similar on the two sides, being small and fast (average duration 60σ), but the knee-jerk on the left was more easily inhibited than in a normal decerebrate preparation, and on the right the inhibition was still more marked. *Labyrinthine* and tonic *neck reflexes* were present on both sides, but were more pronounced on the right than on the left. In each case the resting tension of the muscle was increased by turning the head to the maximal position and decreased by turning the head to the minimal position. The susceptibility to inhibition of the knee-jerk was less when the head was maximal and greater when the head was minimal. The crossed extensor response was also observed to be greater with the head maximal than minimal.

The *crossed extensor reflex* was at first elicited by applying alternating make and break shocks to the contralateral peroneal nerve by moving the camera key with the finger. It was noted that on the left there was a good response which summed readily and relaxed slowly, but on the right the excursion was

not so ample though the character of the response was much the same. Later the crossed extensor response was elicited by applying a tetanizing current from a neon tube oscillator to the contralateral nerve. On the right there was a large well-maintained response which recovered from partial inhibition from a single shock to the ipsilateral peroneal nerve, but on the left the response was very poorly maintained and there was no recovery after inhibition.

Pathological note.—Macroscopical examination of the medulla showed a slight scar of the caudo-lateral surface of the floor of the fourth ventricle, which continued rostralward into the middle cerebellar peduncle. The scar involved the major portion of the vestibular nuclei, especially the lateral nucleus. The restiform body and the brachium conjunctivum were intact. The degenerations in all parts of the cord were similar to those described in Animal II (see fig. 4), with the exception that there was practically no degeneration in the region of the homolateral rubrospinal tract.

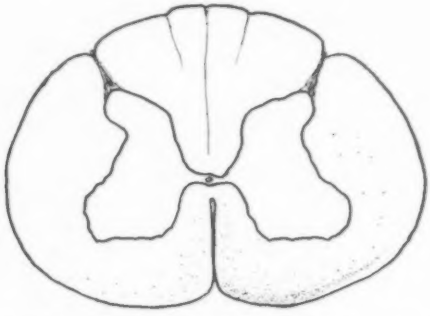

FIG. 4.—Degenerations in Animal V from the cervical level.

CHRONIC EXPERIMENTS. SECTION OF EIGHTH NERVE.

In three cats the left eighth cranial nerve was divided through a cerebellar approach. The protocols of two of these experiments are given in detail below. All of the animals showed symptoms of forced rolling movements to the side of lesion, asymmetrical posture and nystagmus. A fourth symptom deserves mention: for several days after operation the tonus of the limbs was almost equal on the two sides, there being a slight hypotonia of the homolateral limb, but later this changed to a definite *hypertonia* of the homolateral limb which amounted almost to an extensor rigidity in the case of the second animal. In this respect the clinical picture following section of the eighth nerve is exactly opposite to that seen following the destruction of the vestibular nuclei, though in other respects the symptoms from these two lesions are much the same

Animal 1.

Operation. April 29, 1930.—Under "dial" anæsthesia a cerebellar exposure was made and the opening through the occipital bone enlarged to the left with rongeurs. The dura was then freely resected, and a narrow retractor introduced into the left cerebello-pontine angle to retract the lateral lobe of the cerebellum upwards and forwards and the medulla slightly medialwards. By this means a view could be obtained of the eighth nerve just as it emerged from its bony canal. The operation was complicated by slight oozing of blood, but after two attempts the nerve was sectioned with ophthalmological scissors. When hæmostasis was complete the wound was closed in layers.

Post-operative course.—The animal remained deeply narcotized for thirty-six hours, and did not take food or fluid until the fourth day. After that it fed and drank normally and its general condition remained satisfactory. There was a partial paralysis of the left side of the face, apparently due to accidental injury to the seventh cranial nerve at operation. *Forced rolling movements* to the left developed as the anæsthetic wore off, and were very marked for five days, following which they rapidly subsided and disappeared entirely on the ninth day. During the first part of this period attacks of rolling could be induced by a loud noise if the cat was lying quietly, or by sensory stimulation such as rubbing the back. The *posture* was asymmetrical throughout the period of observation, the head being bent to the left, and the chin slightly rotated to the left. This asymmetry was more marked during the first week after operation than later. When picked up by the scruff of the neck the head was bent to the left, the tail to the right, the left fore-limb was extended forward, the right semi-flexed, the left hind-limb was extended backwards, and the right drawn forwards. A slow oscillating *tremor* of the head and shoulders was noted on the eighth day after operation when the animal first attempted to walk. This tremor remained constant during any voluntary activity throughout the remainder of the period of observation. At the end of two weeks the animal was able to crawl along the floor, and at the end of three weeks was able to walk in a crouching position, turning to the left and frequently falling to the left, although it occasionally also fell to the right. There was definitely poor balance, but there was no weakness of the limbs on the side of the lesion, as was noted in the cat with destruction of the vestibular nuclei.

During the first week after operation there were frequent rapid jerks of the eyes to the left with a slow return to the right, although there was no sustained *nystagmus*. After the first week the eye movements were normal. There was slight *hypotonia* of the left fore- and hind-limbs for two or three days following operation, but this decreased and the tone of the limbs became equal. Later the limbs on the left showed a definite hypertonia amounting to spasticity of the left fore-limb at the end of three weeks. The knee-jerks were equal throughout the period of observation. On the last day of observation—the twenty-seventh day after operation—the cat was able to walk, though with considerable difficulty. It crouched close to the floor, frequently falling, more often to the left than to the right. The legs were moved with some uncertainty,

and there was considerable oscillating tremor of the head and shoulders. When held up by the scruff of the neck, the head was bent to the left and the chin slightly rotated towards the left also. The left fore-leg was stiffly extended close to the body, the claws also being extended, the paw slightly flexed. The right fore-limb was semi-flexed. Both hind-limbs hung moderately loose, but the left was slightly more extended than the right, and showed slightly more tone. The toes of the left hind paw were held continuously extended, but were flexed on scratching the pad. On the right the toes were flexed. Knee-jerks and scratch reflex were active and equal. There was no nystagmus.

Myographic examination. May 26, 1930.—Under deep chloroform-ether anæsthesia the preliminary dissection of the hind-limbs was made, after which the cat was decerebrated by intercollicular section. Three hours later the preparation was attached to the myograph in the usual manner, and records were taken from the right and from the left quadriceps femoris. It was noted that when the cat was held symmetrically in the supine position that the resting tension of the fore-limbs was greater on the left than on the right.

There was a good *stretch reflex* on both sides, but it was greater on the right, and on the right there was marked inhibition of the stretch reflex by a single break-shock to the ipsilateral peroneal nerve, whereas on the left there was only slight inhibition. The *knee-jerks* were very fast on both sides, the average duration being 40σ. There was only slight inhibition of the jerk, more marked on the right than on the left.

Neck and labyrinthine reflexes were very marked on the right side, the resting tension of the muscles being much higher with the head in the maximal than in the minimal position. Turning the head had no detectable influence either on the left stretch reflex or upon the inhibition of the jerk.

The *crossed extensor response* on the right, with the head in the maximal position was large, showing a rapid rise with a slow descent and excellent summation. With the head in the neutral position the crossed extensor response was small, and with the head in the minimal position there was no response. On the left with the head maximal, the crossed extensor response was small and subsided slowly. With the head minimal the response was definitely smaller and subsided faster.

Pathological note.—At necropsy the eighth nerve was found to be completely transected. Macroscopical examination of the medulla after fixation in 5 per cent. formalin showed some hæmorrhagic discoloration of the tissue around the point of entrance of the eighth nerve. This discoloration extended 2 or 3 mm. rostralward into the middle cerebellar peduncle.

Animal IV.

Operation. May 8, 1930.—Under "dial" anæsthesia the left eighth cranial nerve was transected in the manner already described.

Post-operative course.—The animal was old and made a very slow recovery from the operation. It was not completely out of the anæsthetic until the

fourth day, and did not take food or fluid until the seventh day, after which it fed normally. The further post-operative course was complicated by a complete left facial paralysis. *Forced rolling movements* developed as the anæsthetic wore off and were very severe for the first eight days, the animal rolling rapidly to the left until it met some obstruction against which it could brace itself. When lying still in the cage these attacks of rolling might be brought on by a loud noise or by stimulation of the skin. During the second week the animal would lie partially on its right side with its legs braced out to the left to prevent its rolling. The rolling movements gradually stopped between the twelfth and fourteenth day. Throughout the period of observation the *posture* was asymmetrical, the head being bent to the left and the chin rotated slightly to the right.

There was a marked spontaneous lateral *nystagmus*, the quick component to the right after operation. This became less during the second week and disappeared during the third week. During the first week after operation there was moderate hypotonia of the left fore- and hind-limbs, but during the second week this changed to a hypertonia of all four limbs, and much more marked on the left side. The *knee-jerks* were at first equally brisk with some fling to the left leg. During the third week, however, the left knee-jerk was more brisk than the right. In the first week the scratch reflex was active on the right but absent on the left. Later, however, the scratch reflex was equal and active on the two sides.

On the last day of observation—the twenty-fifth day after operation—the animal lay quietly in its cage in the horizontal plane with the head and body flexed to the left and the chin rotated slightly to the left. It was able to crawl forward a few steps on the floor in a crouching position, turning and falling to the left. The righting reflexes were active, and were apparently equal on the two sides. When held up by the scruff of the neck the head and tail were flexed to the left, the left fore-limb was stiffly extended closely to the body, the ankle slightly flexed, the claws extended; the left hind-limb was stiffly extended backwards, the toes spread. On scratching the pads of the left foot, however, the toes were flexed. The right fore- and hind-limb were partially flexed. The left knee-jerk was brisker than the right, and the scratch reflex showed a longer latent period on the left than the right.

Myographic examination. June 2, 1930.—Under deep chloroform-ether anæsthesia the same dissection of the hind-limbs was performed as described in the experiments above, and the cat was then decerebrated by intercollicular section through a trephine opening in the skull. Good decerebrate rigidity came on in fifteen to twenty minutes, and was much more marked on the left than on the right side. The left knee-jerk was much more active than the right to inspection. Three hours later the preparation was attached to the myograph. The general condition of the preparation remained good throughout the period of observation. Records were taken from the right and left quadriceps.

Stretch reflex was pronounced on the right, and was markedly inhibited

by a single break-shock to the ipsilateral peroneal nerve. On the left the stretch reflex was not so marked, and there was only very slight, if any, inhibition. The jerks on both sides were small and very fast, averaging 40σ in duration, and there was no inhibition of any of the jerks.

Neck and labyrinthine reflexes were active on the right, the resting tension of the muscle being much higher with the head in the maximal than with the head in the minimal position. On the left, however, there was scarcely any change of the stretch background by changing the position of the head.

The *crossed extensor* response on the right was slight with the head in the maximal position, but absent with the head in the minimal position. On the left side the results were the same, but with even less pronounced differences.

Pathological note.—At necropsy the eighth and seventh nerves of the left side were found to be completely transected, and there were some adhesions to the caudal margin of the left lateral cerebellar lobe, and to the adjoining region of the medulla.

Discussion.

In a previous paper [4] it was shown that the knee-jerk became changed from the decerebrate form to the spinal form when lesions of the homolateral anterior column of the spinal cord were made. The chief descending fibre tracts in this region of a cat's cord are as follows: the tectospinal fasciculus, consisting of both crossed and uncrossed fibres; the reticulospinal fasciculi described by Papez [11], and the vestibulospinal fasciculæ.

It is unlikely that the tecto-spinal system exerts any great influence in the present problem since within quite wide margins the level of decerebration has little effect on the form of the knee-jerk or its susceptibility to inhibition. No definite conclusions, however, can be drawn from the present series of experiments regarding the influence on the knee-jerks of the reticulospinal tracts of Papez. That they play a rôle in the susceptibility to inhibition is probable from the fact that complete section of the homolateral anterior column of the spinal cord has a greater effect on the knee-jerk than a large lesion in the vestibular nuclei in the medulla.

The origin and course of the vestibulospinal tracts have recently been described and discussed by Gray [7] who found a direct and a crossed vestibulospinal tract. The former originates in the lateral nucleus of Deiters and passes caudally in the ventro-lateral column through the entire length of the cord. The latter originates in the descending vestibular nucleus of Schwalbe as the lower arcuate fibres, to cross the midline and descend in the medial longitudinal fasciculus of the opposite side through the entire length of the cord. Both tracts terminate in the ventral gray column. Rasmussen [13] confirmed these

observations, and also found that a number of fibres from the descending vestibular nucleus passed caudally down the cord in the homolateral medial longitudinal fasciculus. As we have shown that only lesions which reach the midline affect the knee-jerk and that lesions of the vestibular nuclei increase the inhibition of the knee-jerk of the contralateral side it is evident that both crossed vestibulo-spinal tracts (which run in the marginal fasciculus) as well as the direct (in the ventrolateral fasciculus) play a rôle in the control of spinal motor centres. It is also evident from the present experiments that the direct tract is the more important of the two.

In investigating the medullary centres influencing the tonus of skeletal muscle, Spiegel [15] found that lesions affecting Deiters', the medial vestibular and the descending vestibular nuclei cause considerable diminution of the rigidity in decerebrate preparations. He also found with lesions which extended more medially and involved the reticular substance of the medulla and the vestibular nuclei that there was complete loss of "tonus" on the homolateral side. All of his experiments, however, were acute, the medullary lesions being made through the posterior approach shortly after rigidity had appeared following decerebration. He concluded that the vestibular nuclei were not solely responsible for the rigidity seen in decerebrate preparations. Further evidence was supplied by the fact that in certain operations the vestibular nuclei of one side were destroyed with diminution of rigidity on the same side, following which the vestibular nuclei of the opposite side were destroyed with return of complete decerebrate rigidity instead of bilateral diminution which might be expected.

Our observations harmonize in general with those of Spiegel, and they also indicate that the lateral vestibular nucleus of Deiters and its descending spinal pathway is undoubtedly the most important of the medullary centres essential for the maintenance of decerebrate rigidity. The results suggest that this nucleus exerts its influence by diminishing the susceptibility to inhibition of the extensor centres in the spinal cord, and we would urge once more [4] that the condition of spinal shock which results when the vestibulospinal pathway is severed is due chiefly to the greatly increased susceptibility to inhibition of the lower spinal centres.

The correlation between the flaccidity observed clinically after a vestibular lesion and the absence of stretch reflex observed myographically is worthy of note, and it bears further evidence of the basic character of the stretch reflex in the maintenance of posture [8], [9].

Section of the eighth nerve causes an augmentation of posture on the same side and the effect is in every respect the opposite of a lesion of the vestibular nucleus. The rigidity and stretch reflexes are increased on the same side and the knee-jerk is more difficult to inhibit.

Summary of Conclusions.

(1) Experimental lessons of the vestibular nuclei in normal animals cause hypotonia on the side of the lesions, forced rolling movements to that side. If decerebrated after an interval of three weeks, the rigidity and stretch reflexes are greatly diminished on the injured side, and the knee-jerk is more readily inhibited. Marchi staining of the cord in such animals has shown extensive degeneration of the homolateral vestibulospinal pathway and slight degeneration in the crossed vestibulospinal tract.

(2) Acute lesions of Deiters' area *after* decerebration also cause great diminution of rigidity on the same side and increased susceptibility of the knee-jerk to inhibition.

(3) We conclude that the nucleus of Deiters and the direct spinal pathway to which it gives rise is chiefly responsible for the differences between the decerebrate and the spinal condition, and hence its integrity is essential for preventing spinal shock.

Addendum.—After our paper had gone to press, we learned that our friend Dr. Paul Martin, of Brussels, had carried out similar experiments and had reached the same conclusions on the modification of the knee-jerk after acute vestibular lesions. Dr. Martin began his experiments six months before ours were undertaken.

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THE ELECTROMYOGRAM OF VOLUNTARY MOVEMENTS IN MAN.

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I.—Electromyograms of Voluntary Movements in which Electrical Effects contributed to by a whole Muscle are recorded with a String-Galvanometer.

Voluntary contraction of a muscle is associated with changes in electrical potential of the tissues immediately surrounding it. These changes have been recorded as deflections of a galvanometer connected to a pair of electrodes placed upon the skin overlying the muscle. In the numerous investigations of these electrical changes or "action currents" as they have been called, it has been the custom to place the electrodes in a line with the long axis of the muscle, and either strapped upon the surface of the skin or penetrating this to make direct electrical contact with the subcutaneous tissues beneath.

The deflections or oscillations will be completely determined if we know their form, their amplitude and their frequency. It is therefore in terms of these three characteristics that they will be described and their variations under differing conditions discussed.

The form of the oscillations.—In the course of his measurements of the latent period of skeletal muscle, Hermann [35] noticed that the electrical changes did not appear simultaneously in all parts of a composite muscle. From a study of their temporal distribution he put forward the idea of a "nervous centre of gravity" of each muscle and described the transverse plane in which this lies as the nervous "equator." His conception, that waves of activity originate in the nervous equator and spread from it up and down the length of the muscle, has been accepted by most subsequent workers, who have agreed with Hoffmann [38] that the electrodes should be placed close together and towards one end of the muscle in order to get deflections of the greatest regularity and amplitude.

Using these conditions, nearly all investigators have found that the oscillating record obtained consists of a series of diphasic deflections, although a few have stated that polyphasic variations in potential are usual (Judin [43]) or constant from certain muscles such as the tibialis anticus (Wacholder [71]). In addition to these comparatively short-lasting oscillating changes in potential, long drawn-out, monophasic shifts of the base line of the record have been described. Most of such effects were originally due to polarization at the surface of the electrodes, but they may also occur when this is avoided and are then due to mechanical shifting of the position of the electrodes on or in the tissues (Gregor and Schilder [31]). Adrian [1] has pointed out that polarization can have no effect upon the form of the quick, phasic oscillations and holds that such mechanical shifting of the electrodes accounts for the maintained alteration in potential which Lewy [48] found to be normally associated with muscular activity.

The amplitude of the oscillations.—Voluntary movements in man give, under the above conditions of recording, electromyograms which do not show deflections of regular amplitude. They stand thus in contrast with reflex movements in man and other vertebrates, and with the normal movements of arthropods (Hoffmann [38], p. 727), Ewald [20]).

In any one record the oscillations usually vary continuously from those of just visible size up to those of maximal size determined by the nature of the electrodes and the sensitivity of the recording apparatus. Although this variation in size is continuous, most observers have described the deflections as falling into two groups: large, primary or "A" waves of lower frequency, and small, secondary or "B" wavelets of higher frequency. Others have carried this differentiation a step

further and have separated three different types, primary and secondary waves, and " Nebenzacken " (Richter [59]). The original view was that the two chief types of deflection were attributable to two different sources or to two different series of phenomena associated with the muscular contraction, and by some this view is still held. Thus Wacholder [72], although agreeing that the graduation is continuous, believes that the difference between the two types, large and small, which may be simultaneously recorded leaves no room for doubt that they are of different origin.

v. Weizsacker [76], in his study of electromyographic changes in certain pathological states, observed that the same degree of contraction of the muscle may be associated with differing records, but that comparison of these records shows that the average amplitude of the oscillations is roughly inversely proportional to their frequency. From this he infers that the total number of electrical impulses coming from the muscle may be constant for any given degree of contraction, and that these impulses will give many small oscillations or a few large ones according to their degree of overlap or fusion. In support of this inference he points out that if during the maintenance of a voluntary effort, which is giving an electromyogram of the normal irregular type, a series of tendon-jerks are elicited in the muscle used, the electromyogram now shows a corresponding series of especially big deflections with silent intervals between.

This view of the deflections as being contributed to by many different parts of the muscle each of which produces electrical changes in its surroundings as it becomes active, and as having varying amplitudes determined by the degree of fusion of the effects of these constituent parts, is now generally accepted. It is clear that this fusion will depend upon the relations of the individual muscle-fibre groups to the electrodes and upon their relative synchronization. When these groups of fibres which are contributing the electrical changes are mainly synchronized and a few only are out of phase, the tendency will be for the records to contain a few large deflections corresponding to the majority in-step, together with many small deflections corresponding to those out of phase or unsynchronized. Many observers have noted the disappearance of the smaller oscillations when regular, larger ones appeared, and Wacholder [70] refers explicitly to the association between the sudden, simultaneous increase of amplitude and of greater synchronization of the innervation impulses to the muscle recorded.

If this view, that differences in amplitude of the deflections are due

to different degrees of synchronization of the muscle-fibres, be correct then the amplitude cannot be expected to give any measure of the force of the contraction. This conclusion is supported by Adrian [1] and Wacholder [74] in their reviews of this subject.

A discussion of the variations in amplitude which have been described can, for these reasons, be more usefully postponed until the question of innervation frequency is considered.

The frequency of the oscillations.—Comparison of the figures given by different observers for the deflection frequency is complicated by three factors :—

(1) Some observers have counted the waves of different size-groups separately—others the total number of waves irrespective of size.

(2) Differences in galvanometer sensitivity lead to great differences in the appearance of the records obtained. Where total deflections are in question, this leads to an apparent reduction only in frequency of the less sensitive records, due to the fact that some of the deflections are too small to be noticed. Where different size-groups are counted separately, it leads to a relative diminution of the frequency of the group of larger deflections when the sensitivity is decreased.

(3) In connection with few records have the conditions of recording been adequately described—in particular in respect of the tension developed by the muscle at the time of recording.

In 1912 Piper [49] published his conclusion that the frequency of the large waves alone was 50 per second for all voluntary movements and was unaltered for different strengths of contraction. Subsequent workers have agreed that this rate is very often seen in the electromyograms of voluntary movements, more especially when the muscular effort is a large one, but that it may also be much lower. With increasing sensitivity of recording and with electrodes which are influenced by more strictly localized changes only, rates as low as 8-10 (Wacholder [74]) and 5-8 per second (Richter [59]) have been found associated with slight degrees of voluntary effort. Even when the greatest effort is made the rate has not been found to rise above 75 per second, and most observers give figures which suggest that the most common frequency for the large waves lies in the neighbourhood of Piper's figures. (Altenburger [3], 50-60 ; Bornstein and Sanger [11], 50 ; Gregor and Schilder [31], 37-70 ; Richter [59], 40-65 ; Wacholder [71], 50-75 ; Henriques and Lindhard [33], 40-65 ; Salamonson [61], 50 ; Proebster [53], 40 ; Cobb and Forbes [13], 40-58 ; Semereau and Weiler [64], 50.) The significance

of this 50 per second rate has yet to be determined and will be discussed later.

The results of those investigators who have estimated the frequency of the smaller waves are still less comparable, because while with stronger voluntary effort the large waves tend to become more regular, the smaller waves become irregular and grouped, or disappear. With moderate degrees of contraction their rate has been variously estimated at from 50 to 300 per second. (Altenburger [3], 160-190; Wacholder [71], 150-180; Richter [59], 180-250; Proebster [53], 200; Pradwicz Neminski [57], 20-150; Trendelenburg and Schutz [68], 120.)

We are here considering the frequency of galvanometer-deflections, and in the light of more recent knowledge of the electrical field set up by active muscle and of the varying synchronism of innervation of different muscle-fibre groups, it is clear that no significance attaches to these figures. They could only have value if some assumption such as that made by Athanasiu [5] were correct, i.e., that the total muscle mass is always synchronously innervated. In this case also, the same author's suggestion that the ratio of the small to the large wave frequency tends to be constant (4 : 1) would be not so improbable. Since we have good grounds for supposing this suggestion to be invalid, such frequency measurements and their comparison are probably meaningless.

To summarize the evidence of the above paragraphs, we may state that the electromyogram of normal voluntary movements, as recorded by means of a string-galvanometer and surface electrodes, shows a series of diphasic deflections or oscillations. These are of irregular amplitude but fall roughly into two groups, large and small deflections, and they recur at the average rate of 50 per second and 180 per second respectively. With increasing muscle tension the galvanometer deflections show a rise in total frequency, an increase in average amplitude and no change in their individual form. These are only generalizations, and neither of the variables, frequency and amplitude, alter at any stage proportionately with the rise in tension. Moreover, a given tension in the same muscle repeatedly and similarly recorded, is not invariably associated with the same type of electromyogram. Thus it has been shown by Wacholder [70] that contraction and relaxation respectively, through a certain phase of muscle tension, yield quite different pictures, and that the same muscle used as an agonist, an antagonist, or as a synergist for different movements, gives a different record in each case.

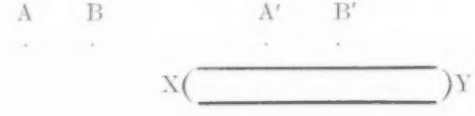
The interpretation of these differences depends upon differences in the innervation and not in the forms of muscular activity, and the

correctness with which we can infer the character of this innervation from the galvanometer deflections will be decided by our ability to determine the source of the potential differences recorded and the factors governing their appearance and form.

II.—The Electrical Field set up in the Surroundings of a Contracting Muscle.

A muscle strip isolated from all other structures and stimulated to contract shows a series of changes in electrical potential, "action currents," along its surface as the wave of mechanical activity spreads through it. In the light of the results previously obtained from the use of such isolated muscle preparations, it has proved difficult to interpret the galvanometric oscillations associated with the activity of intact muscles when electrodes are placed upon the surface of the body. Recently, however, the work of Craib has made this interpretation possible. He has shown [16] that the deflections resulting from activity of an isolated muscle preparation depend upon the amount and distribution of the conducting medium surrounding the muscle and upon the position of the electrodes within this medium in which is situated the electrical field. The records obtained from a muscle strip in air differ greatly from those given by the same strip when surrounded with saline solution, and Craib has studied the galvanometric responses obtained from electrodes placed in different parts of such a conducting medium. His observations have proved successful in the interpretation of the changes in electrical potential which accompany the activity of cardiac muscle, and we may look to them to provide a satisfactory interpretation of the electromyogram of voluntary contraction in skeletal muscle.

It is only comparatively recently that needle electrodes thrust right into the active muscle have been employed (Adrian and Bronk [2]). As has been pointed out, the electromyograms of voluntary movements previously published have been obtained by the use of electrodes placed either upon the surface of, or just beneath, the skin. It is with the interpretation of such electromyograms that we are now concerned, i.e., those recorded from what we may speak of as "distant" electrodes arranged parallel to the long axis of the muscle and towards one of its ends.



If in the above figure X—Y be taken to represent the active portion of the intact muscle and A—B, A′—B′, A″—B″, pairs of electrodes making contact with the surrounding tissues, we learn from Craib's work that as a wave of activity passes from X to Y a diphasic deflection will be recorded from A—B and A″—B″ and a polyphasic deflection from A′—B′. This last deflection will consist typically of six phases, three contributed by the passage of the active region beneath A′ and three by the passage beneath B′. These six phases are not of equal magnitude and they suffer various degrees of fusion which are dependent upon the length of the active region, the nearness of A′ to B′ and of A′—B′ to the muscle, and the nearness of either A′ or B′ to one end of the muscle. As the distance of the electrodes from the muscle is increased all the phases are diminished and the already smaller elements of the deflection rapidly become too small to record. As a result of the overlapping of the effects from A′ and B′, and of the dropping out of phases which have become too small to record, the deflections usually obtained from such muscle preparations with electrodes in the position of A′ and B′ are triphasic when the electrodes are near to the muscle and diphasic when they are remote.

From these results we see that if we regard the strip X—Y as consisting of the active skeletal muscle, and the electrical field resulting from its activity as being set up in the surrounding conducting medium provided by the other structures of the limb in which this muscle is lying *in situ*, we may summarize concerning the deflections to be expected in this way: the deflections will be diphasic in form in all cases except where the electrodes are placed in intimate contact with the muscle, are well separated each from the other and from the ends of the muscle, and are in such a position that the wave of contraction or activity passes immediately beneath them both: in which case they will be triphasic.

In the section dealing with the form of the deflections making up the voluntary electromyogram it was stated (p. 345) that Wacholder had found triphasic deflections to be constant for the tibialis anticus. It is possible that the relatively superficial position of this muscle, which allows surface electrodes to come into closer contact with it than with most other muscles recorded, is sufficient to explain this result.

The results above described have been obtained from a parallel-fibred muscle strip, stimulated to contract at one end, so that a wave of contraction passes along the muscle from the end stimulated. In the case of voluntary contractions of intact skeletal muscles we are

dealing, for the most part, with muscles which have in some degree a pennate arrangement of their fibres and in which the spread of the contraction is not entirely a function of muscular conduction but is probably the result of the intramuscular nerve filaments. In the case of the isolated muscle strip the moment at which any particular part of the muscle contracts depends upon its distance from the end stimulated. In the case of the intact, composite skeletal muscle it might be expected that the sequence in which the different parts of the muscle contract depends upon their physical distribution about the tendon through which their effect is exerted, and not upon their distance from any particular transverse plane of the muscle, e.g., the nervous equator.

That some parts of a muscle such as the quadriceps femoris are normally thrown into contraction before others has been shown by Golla and Cook [30], who find that when this muscle is voluntarily contracted a wave of activation seems to spread down the muscle distally. That this effect is not due to conduction in the intramuscular nervous filaments is considered by them as decided by the observation that the rate of spread is six times as slow as the rate of nerve conduction.

These authors find that when the two electrodes are either both proximal or both distal to the nervous equator, the current of action is diphasic and in an opposite direction in the two cases. When the electrodes are placed one on either side of the equator the current of action is triphasic, as a result, they believe, of "the interference of two diphasic electrical responses travelling in opposite directions" (p. 50). This interpretation is not tenable. For presumably in the other cases of the two electrodes placed toward one end of the muscle so that activity spreads first under one and then under the other, the diphasic form of the action current is regarded as being due to relative negativity of the first electrode with regard to the second, and then of the second with regard to the first. If it were other than this one would expect a polyphasic deflection. When the electrodes are placed one on either side of the nervous equator, on the supposition that a wave of contraction (negativity) spreads up and down the muscle from this plane, the effect of the wave spreading proximally should be a transitory relative negativity of the proximal electrode, that of the wave spreading distally should be transitory relative negativity of the distal electrode. We should thus expect the electromyogram to show a diphasic alteration in relative potential as a result of the fusion of these two monophasic changes. In no case could a triphasic current of action be anticipated. However, these results are capable of another explanation. It has been

shown above that the conditions which favour the appearance of triphasic action currents include wide separation of the electrodes from each other and from the ends of the muscle, so that the active region lies wholly beneath them and not mainly to one or the other side. These conditions are more nearly fulfilled by the case in which the electrodes are separated to either side of the nervous equator than in any other position, and it is suggested that this, rather than the presence of a dual wave of activity, is the cause of the triphasic response so obtained.

III.—The Relation between the Electrical and Mechanical Effects in Muscle.

Of the three associated phenomena which commonly characterize the activity of muscles—namely, shortening or tension development, metabolism with heat production, and electrical phenomena—it is the first which gives them their value in the intact organism and which may properly be regarded as their function. It is thus in terms of mechanical change that the galvanometer deflections must be considered. The electrical variations responsible for these deflections occur only in intact muscle in association with activity, i.e., shortening or tension development. Whether the three phenomena mentioned above are all concomitant expressions of some underlying fundamental change, or whether any one of them stands in casual relationship to the others, will have in due course to be considered.

That the electrical variations observed in the locality of a contracting muscle are not adventitious to the muscle function in the same sense as is the heat liberated on contraction, is indicated by the constancy with which apparently normal mechanical, metabolic and electrical changes are set up in response to electrical stimulation of the denervated muscle. We may now consider the temporal and quantitative relationships of the electrical and mechanical changes.

The relation in time.—Einthoven and Hugenholtz [19], finding that there was no interval between the appearance of the electrical and of the mechanical change, concluded that there were but different results of the same process. A similar conclusion was reached by Arbeiter. Fulton has repeated these observations with more exact methods and finds that the electrical precedes the mechanical change in the skeletal muscle of the frog by about 1·5 sigma [27]. A much longer value is given by Kleinknecht [46] who reports that cooling of the muscle may increase the period up to three times its original value. Fulton has also

found that the size of the electrical change is proportional to the initial heat liberated. These results indicate that the electrical change precedes the mechanical change by an interval which suggests that it is an index of those processes of which the tension contraction effect is an immediate result.

In the case of the ordinary phasic twitches of skeletal muscle the electrical is of much shorter duration than the mechanical effect, and while in tetanus a plateau of maintained tension may result from complete fusion of the mechanical contractions, the electrical responses to stimulation always remain distinct and separate. It is only in the long drawn-out action currents associated with some forms of tonic contracture in striated muscle that any approximation of duration of electrical and mechanical responses can be found (Lee [47], Bishop and Gilson [10]).

The relative size of electrical and mechanical responses.—In a parallel-fibred muscle increase of submaximal stimuli leads to increase in size of both effects, due to the activation of more and more muscle fibres. When maximal tension development is reached further increase in the strength of the stimulus does not lead to any further increase in amplitude of the electrical variation. With repeated just-maximal stimuli both effects diminish as fatigue appears. Fulton finds [25] that they run parallel courses in their decrease to become extinct together. Beritoff [9] on the other hand finds that neither their courses to extinction nor their recovery rates after cessation of stimulation are parallel. But he used the gastrocnemius preparation from a frog, and Forbes, Ray and Hopkins [22] have explained the prior extinction of the mechanical effect found by him in terms of the obliquity of the fibres in this muscle.

In the unfatigued muscle-fibre it is to be expected that the electrical effect will be of constant amplitude and that it will give a true indication of the functional mechanical activity of that fibre however this is stimulated, e.g., voluntarily or reflexly.

IV.—The Relation of Galvanometer Deflections to Activity of the Central Nervous System.

As we have seen, the familiar electromyogram of voluntary muscular contractions obtained by the use of two distant electrodes and a string-galvanometer consists of a number of deflections of varying sizes and differing rates. But, as Hoffmann [38] has pointed out, we cannot safely infer anything as to concomitant central nervous activities from

records of electrical phenomena in muscle obtained in this way. It is true, as will appear later, that records from electrodes arranged so as to "tap" individual muscle fibres do provide information as to the conditions of motor-nerve innervation and thus allow of conclusions as to the activity of nerve centres. For the present, however, we are concerned with the older type of electromyogram obtained by distant electrodes which tap a whole muscle mass.

In Chapter III, reasons were advanced for believing that the electrical variations under consideration were entirely an expression of muscular activity, and there are no grounds for suggesting that deflections of one size and rhythm are contributed by nervous impulses of a corresponding rhythm, and that those of another rate are arising in the muscular responses to those impulses. All arise in the muscle and thus all stand in the same relation to the activity of the motor-nerve neurones. The size of each deflection will depend mainly upon the number of muscle units engaged.

Two assumptions have been made with regard to the relation between the frequency of motor-nerve impulses and the galvanometer deflection rate. One assumption, that the muscle possesses an inherent rhythm at which it responds to motor-nerve impulses at a higher rate, has been carefully examined and found insufficient by Adrian [1]. The other, that the muscle shows a repetitive response to a slower rate of innervation, is based upon equally unsatisfactory grounds (Wacholder).

Although the result of experiments designed to show simultaneity of activation of different parts of the same muscle (Wacholder) or of neighbouring muscles (Altenburger and Forster) must be taken with reserve in the absence of strict control as to the origin of such action currents, simultaneous records of motor-nerve impulses and muscular contraction (as taken from the phrenic nerve and diaphragm by Dittler and Garten [17]) give strong support to the view above stated that the muscle contributes nothing in the way of inherent rhythm to the frequency of the deflections.

Since the amplitude of the galvanometer deflection is determined by the number of muscle units active, if each unit were innervated by a separate and distinct path from the central nervous system we should be able to decide from the frequency and amplitude alone just how many lower motor neurones were active at any given moment and at what rate. This is, however, not the case. One motor neurone may activate several muscle units, or a single muscle unit may derive its innervation from more than one motor unit. The second of these possibilities has

been shown to be unlikely in the case of the frog and the rabbit (Fulton [26]), for which animals it was originally suggested, and it is improbable that it occurs in man. The former suggestion is almost certainly true. The number of nerve-fibres in a motor nerve is far smaller than the number of muscle fibres in the muscle which it innervates, so that it is clear that each nerve-fibre must be capable of activating more than one muscle-fibre, in much the same way as Adrian has shown to hold for the afferent nerves from skeletal muscle where several tendon end-organs are connected with each afferent fibre. If we could assume further that activity of a nerve-fibre always entails contraction of all the muscle-fibres which it innervates then this complication could be disregarded and the units of electrical field production shifted from the individual fibre to the composite unit. But it will be later shown that such an assumption is not valid.

The central nervous discharge may be varied only in certain ways, e.g., in respect of the number of neurones involved, the rate of the impulses in each neurone, and the time relations of the activities of the various neurones.

In view of the relatively short refractory period of the peripheral nerves it is probable that they do not contribute anything in the nature of an inherent rhythm modifying that of the central motor innervation : but that they simply pass on unaltered the train of impulses which results from voluntary or reflex excitation of the anterior horn-cells. It is known that repeated rhythmic stimulation of an afferent nerve will lead to reflex motor discharges which reproduce faithfully the stimulating rhythm up to rates higher than those which are met in the normal electromyogram, so that it is probable that the source of any rhythms present during voluntary activation must be sought at a higher level than the lower motor neurones. The results of rhythmic stimulation of the cortical motor centres have not given unequivocal evidence in favour either of a central or of a peripheral dominant rhythm. Keller [44] found that the rate of stimulation of the cortex was followed strictly by the electrical discharge from the muscle up to 170 stimuli per second. Cooper and Denny Brown [15] working with monkeys found a similar parallelism from 160 to 180 per second, and Plattner [50] found it in dogs between 80 to 200 per second. However, Hoffmann [39] found that in dogs only rates between 40 and 50 per second were accurately followed, at lower rates the muscle responded at a double or treble frequency and at higher rates the frequency was halved. The first experiments suggest that if there is a dominant rhythm within the

limits given then it is probably not cortical in origin, the second suggests that the cortex tends normally to discharge at 50 per second, a rate which has been much in favour as a characteristic of normal voluntary electromyograms.

It is of interest here to examine the possible significance of this 50 per second rhythm. It has been subscribed to by so many observers and confirmed in so many different ways that it cannot be due to any particular form of recording, but must represent the tendency for periodic large alterations in the electric field at this rate, i.e., for a tendency to synchronization of the muscle elements at this rate. That it is a synchronization effect follows clearly from the fact that it is only

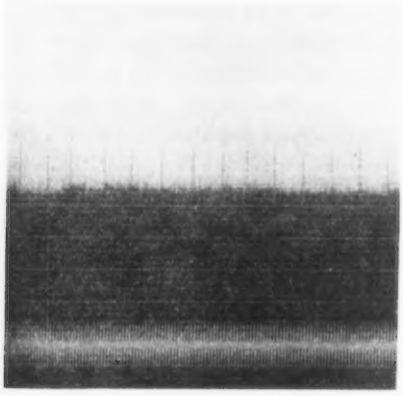


FIG. 1.

by synchronization of the smaller elements that larger deflections can be produced and also from the fact that in those records or in those parts of any one record in which this rhythm is best marked the secondary waves due to elements out of phase are least conspicuous.

From a muscle such as the triceps of the arm during voluntary activation, using needle electrodes (Adrian and Bronk [2]) and the Matthews oscillograph, records such as those shown in fig. 1 are obtainable. With the slightest degree of voluntary effort a series of deflections of equal and small amplitude are obtained; and evidence has been given elsewhere (Pritchard [52]) for regarding these as coming from the individual muscle units. With higher degrees of effort there is found not only an increase of rate but an increase of amplitude: the intermediate stages of fusion of individual units can be clearly traced

upon intermediate records. If the record of a strong voluntary contraction is measured it is found that the larger deflections, which are quite irregularly spaced, occur at the rate of 45 to 48 per second, upon a background of very many smaller deflections, these deflections are due to the same periodic electrical changes which give the large deflections —"A" or primary waves—of the string-galvanometer record and Piper's "50er Rhythmus." It was suggested in the same paper that the maximal number of motor units which could be simultaneously activated during a voluntary effort might be a function of the neuronic connections "either in the peripheral nervous pathway or at a higher level."

To summarize this view of the relation between electromyographic deflections and central nervous discharge, it is suggested that the type of record obtained with the string-galvanometer and two independent distant electrodes gives no direct clue to the rate of motor impulses in the peripheral nerve-fibres, in view of the complexity of the muscle mass, all of which contributes to an electrical field which is "tapped" in comparatively distant regions. Thus it is impossible to separate the responses obtained from functional units one from the other, but the rates of deflection so obtained are determined wholly by the rates of discharge along the lower motor neurones. Records obtained from more strictly localized intramuscular electrodes do enable us to infer with a high degree of probability the rates of discharge in certain cases. The 50 per second rhythm, which has been held to characterize the electromyograms of voluntary movements and for which many different origins have been suggested, is probably a function of the relation between the number of motor-fibres and the number of muscle units which they supply, and does not represent the inherent tendency to discharge at that rate either of the muscle or of any part of the central nervous mechanism.

V.—The Influence of Fatigue upon the Action Currents of Voluntary Movements.

Three changes have been recorded affecting respectively the amplitude, the form and the frequency of rhythm of the galvanometer deflections. The fall in total frequency of the large waves from 50 per second to 35 or even 20 per second noted by Piper, has been generally confirmed (Altenburger [3], Altenburger and Guttmann [4], Gregor and Schilder [31], Cobb and Forbes [13]). An increase in frequency has not been observed, but some (Dittler and Garten [17]) who have counted the total oscillations and not the larger waves alone,

have come to the conclusion that the diminution in frequency of the former is apparent only, and is due to the fact that some oscillations become too small to be detected.

In almost all cases the observed drop in frequency is associated with an increase in amplitude of the larger waves. Altenburger and Guttmann [4] have established from their records a reciprocal relation between frequency and amplitude as holding good in the earlier stages of slowly fatigued voluntary movements. The interpretation of this relationship is helped by the observation that associated with it there is a diminution or absence of the "Nebenzacken" or secondary wavelets leading to relative quiescence of the galvanometer between the larger waves (Piper [49], Gregor and Schilder [31], Altenburger [3]). This suggests that with the onset of fatigue the motor cells of the cord tend to fire off simultaneously in volleys so that there is an increasing degree of synchronization of the different muscle units: a conclusion reached by Bornstein and Sanger [11], Fulton [24] and Haas [34]. The further observation of some workers (Piper [49], Altenburger [3]), that the large waves are more drawn out, i.e., of increased duration—although this is not found by others (Cobb and Forbes [13], Gregor and Schilder [31])—may be explained as due to the incomplete volleying of the neuromotor units giving incomplete fusion of the muscle units, whose slight temporal dispersion leads to a prolongation of the summated electrical action currents.

Altenburger [3] and Altenburger and Guttmann [4] have distinguished between changes occurring in loaded and in unloaded muscle and in fatigue attained rapidly or gradually. Thus they found that the increase in amplitude of the oscillations is found only in loaded muscle and that late in fatigue it gives way to an increase in duration, the synchronization of different muscle elements becoming less perfect. When the volleying of impulses becomes still more complete, or when a further stage of approximation of the moments of discharge of greater numbers of neuromotor units is reached so that the summated effects from different groups of muscle units tend to be thrown together, the galvanometer record shows grouping of the deflections corresponding with periodic outbursts of approximately synchronized activity. Bornstein and Sanger [11] have associated this grouping or volleying of higher degrees of fatigue with the development of fatigue tremor. The greater the degree of fusion or volleying of the impulses the larger will be the number of muscle units which become active simultaneously and the more pronounced will be the tremor.

It appears, therefore, that when voluntary activity of the same muscle is prolonged there develops a tendency for volleying of the motor discharge with increased synchronization of the different muscle units, leading to action currents of greater amplitude and diminished frequency, and finally to a degree of fusion which results in tremulous maintenance of tension by the whole muscle.

These being the changes in the electromyogram in fatigue it is natural to inquire as to the mechanism of their production. About this question there is at present less agreement. It has been shown (Pritchard [52]) that the volleying of impulses during fatigue of voluntary contractions is exactly similar to that which may be seen to characterize any severe muscular effort which leads to a tremulous contraction; and this from the outset of such an effort and before the normal fatigue mechanism can be regarded as playing any part. It is, therefore, reasonable to look within the central nervous system for the mechanism productive of both fatigue and effort tremor. The changes occurring in the muscle itself during fatigue do not provide an explanation. It is probable that the muscle mechanisms have a sufficient reserve to render them relatively unfatigued under physiological conditions, including those of voluntary movement fatigue (Guerra [32]). It has been shown (Beritoff [9]) that when a muscle is fatigued experimentally by repeated stimulation of its motor nerve the current of action is affected less easily and to a smaller extent than the power of contraction—indicating a relatively greater resistance to fatigue which is paralleled by the more rapid and earlier complete recovery of the action currents after cessation of the motor-nerve stimuli. Beritoff explains this effect as due to the fact that the electrical effect is associated with excitability ("erregbar Substanz") and not with the acid breakdown which results in the mechanical change. Fulton [26], who holds that action current and contraction run a parallel course towards extinction during fatigue, points out that since the accumulation of contraction catabolites in the muscle results in diminution of the action current, the increase in amplitude of the oscillations seen in the fatigue electromyogram cannot be referred to an increase of electrical effect from each muscle unit active, but can only be explained on a basis of synchronism of their innervation.

v. Weizsacker [76] refers the diminution in frequency and increase in amplitude to fatigue of the proprioceptive nerve-endings in the muscle, holding that the central nervous system tends to discharge from its motor neurones with a slow automatic rhythm, and that in all normal

voluntary movements this rhythm is broken up by the interference of proprioceptive impulses received from the muscle innervated. With fatigue of these proprioceptive nerve-endings and the consequent diminution of their effect, there ensues a nearer approximation to the slower automatic rhythm of the central nervous system. Against the suggestion that such changes may result from a fatigue of the central nervous discharge, he draws attention to the results of Hoffmann [39] who found no evidence of fatigue in the stretch reflexes after 10,000 repetitions in three minutes. But this provides an equally sound reason for rejecting the notion of fatigue of the proprioceptive nerve-endings, since the integrity of the stretch reflex is as much dependent upon their co-operation as it is upon the motor discharge.

The increase of the tendon-jerks normally associated with voluntary fatigue indicates that the proprioceptive stretch reflex mechanism is made hyper-excitable rather than diminished in activity and accords with Fulton's suggestion that an accumulation of catabolites in the muscles may make the proprioceptive nerve-endings more readily responsive. This hyper-activity of the proprioceptive reflex mechanism will give a relative increase in the reflex component of voluntary innervation and a relative diminution of the cortical component leading to diminished "temporal dispersion" (Fulton [24]), i.e., increased synchronism, increased amplitude of the action currents, and mechanical tremor. The observation that the rates of clonus and of fatigue tremor are identical is adduced in support of this view. It has been shown that a very strong isometric voluntary contraction of a group of muscles is frequently tremulous from its beginning and the electromyogram shows a characteristic tremor; this suggests that the majority of the effective muscle units have been thrown into synchronous activity. When the same group of muscles is fatigued by repeated voluntary contraction with a very light load (the muscles being allowed to shorten and extend fully with each effort), shortly after fatigue is noticed there appear changes in the myographic and electromyographic records which are strictly comparable to the above, i.e., shortening and lengthening become tremulous and intermittent and the electrical discharge becomes volleyed in the same way. There can be no adequate reason for regarding the changes occurring in these two sets of circumstances as being different in origin or nature, and the terms "effort tremor" and "effort syndrome" may be used in respect of either. They both have a sense of strenuous activity as the mental counterpart of the physical changes, and it has been pointed out in the same paper (Pritchard [52]) that

the tremulous movements of paralysis agitans are indistinguishable from those of isotonic and isometric fatigue in respect of all three characteristics.

It is convenient to have some term which will express the occurrence of this group of phenomena modifying voluntary movements. The term *effort syndrome* has been here suggested and will be used to denote that type or that phase of voluntary movements, accompanied by a sense of (strenuous) effort, in which the mechanical shortening is intermittent or the tension maintenance discontinuous, and the electromyogram is of the grouped or volleyed type. The group frequency of the electromyogram is in any one case identical with the intermittent frequency of the mechanical contraction, the one being a record of periodic outbursts of activity which determine the occurrence of the other.

In the case of repeated isotonic movements and of paralysis agitans, induced fatigue in the former and chronic fatigue in the latter might lead to fatigue of the peripheral mechanisms of contraction and to the accumulation of catabolites which exercise a secondary effect upon the proprioceptive nerve-endings. But such a sequence of events can clearly not be held responsible for the alterations in the efferent discharge producing the effort syndrome in the case of a suddenly initiated, strong isometric movement.

Again, the excessive stimulation of the proprioceptive end-organs which probably occurs in a very strong isometric contraction might be regarded as providing an explanation for the effort syndrome in this case, but the development of an excessive tension does not take place in the case of unloaded shortening and lengthening movements and, therefore, cannot account for the appearance of the effort syndrome in either isotonic fatigue or in paralysis agitans. Further, it has been shown by Adrian [1] that such excessive stimulation of the tendon organs leads to their well-marked fatigue with diminution in their rate of discharge. We have seen above that there are serious objections to the notion of fatigue of proprioceptive mechanism as underlying these synchronization effects and that their hyper-activity would provide a better explanation.

We must, therefore, conclude that there is no adequate explanation of the voluntary effort syndrome in terms of peripheral changes such as an alteration in the muscle or in the afferent impulses received from it. It remains to be determined whether there is any explanation forthcoming from consideration of the central nervous mechanism on the purely motor side.

The change in the form of the electromyogram which results from the fusion of asynchronous elements into synchronous groups is clear cut. It results in a series of separate deflections of uniform amplitude occurring at the rate of 45 to 48 per second. It is due to the use, by the innervation mechanism, of a new effector unit; the composite group innervated from a single or from intimately related motor neurones.

The further change, occurring in fatigue, which results in the grouping together of these larger deflections is by contrast the consequence of a process which is not nearly so definite: the groups, as such, are distinct but the further fusion of deflections is never complete. The individual deflections, although grouped, still occur at the same average rate. These characteristics suggest that the grouping or volleying effect is the result of some periodicity of action of a central multi-fibre mechanism having a natural frequency which is only elicited under abnormal conditions.

The mechanism which has been shown to account satisfactorily for the first stage of synchronization of the motor impulses, i.e., that from individual units to composite units, may be operative wherever any single proximal centrifugal pathway exerts control over several distal centrifugal paths. Since this condition obtains at higher levels in the nervous system it might be expected that the further degree of approximate synchronism of the larger units results from a similar mechanism higher up the motor paths. It might for example take place at the level of transmission of motor innervation from the pyramidal fibres to the anterior horn cells, or at the level of influence of the cells of the motor cortex by some other region of the cerebrum. There are, however, several reasons why it is improbable that such a mechanism comes into play at these levels.

In the first place, the two steps towards temporal fusion of deflections, as above indicated, are of different types. The peripheral mechanism (a function of the lower motor neurone and muscle-fibre) is one frequently called into play in all movements of moderate intensity. It results in a type of innervation which is still completely dispersed and aperiodic, giving still a smooth contraction and sustained tension development. The suggested central mechanism is one which provides a type of action which characterizes abnormal efforts, whether in disease, fatigue, or over-exertion. It results in a form of discharge which is uneconomical; the contraction is intermittent and the tension discontinuous. In normal persons the primary synchronism of individual units into larger units invariably precedes the secondary synchronism

into groups. For this to obtain the frequency of the impulses in the most central paths requisite to synchronize all the intermediate paths must be greater than that sufficient for synchronizing all the individual muscle-fibres into a secondary muscle unit. Therefore, if the central and peripheral mechanisms are of identical type, the first stage of fusion into larger muscle units must always precede the second stage of grouping into volleys. But this is not the case in either isotonic fatigue or in paralysis agitans, where volleying and tremor can take place with the motor units still acting separately.

The groups of deflections in the effort syndrome indicate outbursts of activity producing a tremulous contraction which involves the whole or most of the muscle, so that for the grouping to be the result of a mechanism similar to that already outlined would take us up to a level at which a single neurone, or a multifibre but synchronous motor pathway is at work providing all the excitatory innervation. Such tremor cannot be contributed by anything less than the whole motor mechanism at some level. It must mean that at such a level the mechanism, although still composite with regard to the number of pathways through it, is imposing upon the impulses which it transmits a secondary regular rhythm. It must be a mechanism which is capable of responding aperiodically in normal circumstances, but which under certain conditions contributes that secondary rhythm which results in its breaking up a continuous stream of impulses coming to it into a discontinuous stream of impulses passing from it, i.e., in converting a steady incoming stream of impulses into an intermittent outflow.

In the field of applied physics there are several other similarly related systems which show the same type of action, i.e., the conversion of a continuous into a discontinuous discharge by virtue of an inherent periodicity, and it is instructive to examine these further.

The ordinary whistle when blown into gently transmits merely a rush of air; as the speed with which the air passes through is increased a value is reached at which a note is elicited, i.e., at a certain pressure of air the natural period of the whistle is excited and oscillations are set up. Perhaps more strictly parallel if less familiar is the behaviour of fluid flowing through a circular pipe. At low velocities the lines of flow are axial, that part of the fluid flowing down the centre of the pipe having the greatest velocity and that in contact with the walls being virtually at rest. As the pressure of input and speed of flow are increased, a value is reached at which the above conditions are no longer present. Vortex flow is substituted for axial flow; the fluid rolls

over the sides of the pipe. Vortex waves are set up and the discharge becomes oscillating. The rate of flow at which this takes place depends upon the pipe as well as upon the fluid, and has been empirically expressed in terms of the viscosity and density of the fluid and the radius of the pipe.

An example of an oscillating system in another field of work is provided by an electric circuit which contains a condenser and an inductance; the frequency of oscillation of such a system may be shown to be a function of the capacity of the condenser and of the inductance.

It may be argued that in these examples the parallel is imperfect because the input is truly continuous, whereas this is never the case with central nervous innervation. Yet if we examine other systems to which the objection cannot be raised, we find that the same type of relation between the critical rate of input and the properties of the system holds good. Thus if a stretched wire is periodically displaced transversely at one point and as often released, a series of waves travel along the wire in both directions from the point displaced, these waves having a distance apart dependent upon the frequency of displacement. As this frequency rate is progressively increased a stage is reached at which the natural frequency of the wire is called out and stable oscillations of a different form are set up. This frequency is expressed in terms of the tension in the wire and the mass per unit length. The The above vibrations are transverse. Longitudinal vibrations similarly set up in any suitable medium such as a gas find a similar frequency in terms of the modulus of elasticity, and the density of the medium.

Taking each of these examples of oscillatory system, we find that tho essential properties upon which its characteristics depend, and in terms of which that critical input value which elicits an oscillatory discharge is expressed, are two in number and invariably of the same physical dimensions. One property is that of inertia or akin to it, and the other of elasticity, i.e., one limits the immediate effect of an impulsive stimulus or disturbing force and the other tends constantly to restore the condition of quiescence or equilibrium.

Thus in the electric system the inductance corresponds to the lag or inertia and the capacity to the elasticity. In the fluid system the viscosity represents the inertia, and the density the resistance to compression or tendency to re-expansion. In the stretched wire the mass per unit length determines the inertia which limits displacement and contributes the momentum of overthrow, and the tension in the wire

determines the restoring force pulling the displaced elements back into line.

These two properties, one akin to inertia and the other to elasticity, endow the system in which they occur with the oscillatory characteristics which it shows. If, therefore, the central nervous mechanism which mediates the motor discharge between the cortex and the muscle, proves on closer examination to behave in its other activities as well, in such a way as to lead us to ascribe to it properties having similar characteristics as inertia or momentum and as elasticity, we may conclude that it contains within itself those properties the combination of which is both essential and sufficient for the production of an oscillating or volleyed discharge.

Such a mechanism cannot be determined by the physical property of the muscle itself because this varies so considerably in the different conditions in which the tremor effect can be observed. It is probably not at the level of the cortex because, as Fulton has pointed out, the rate of effort tremor and of clonus are identical under comparable conditions, so that it is probably a mechanism which is shared by both voluntary and proprioceptive reflex activities. It cannot be anywhere along the lower motor neurones because the whole muscle is involved. We must conclude, therefore, that if it exists at all it must be somewhere along the course of the reflex pathway central to the motor neurones and yet on the efferent side of the reflex arc.

In a subsequent paper it will be argued that a mechanism having the properties of mass and elasticity and situated in this position will adequately account for the observed facts of spinal reflex behaviour in respect of excitation and inhibition. These facts have been examined and their interpretation attempted in terms of such a "ballistic model." The conclusion is reached that not only is the behaviour of the effector mechanism, under conditions of reflex excitation, also compatible with the presence of a nervous centre acting in a certain way, paralleled in the field of mechanics by a system having these properties, but that it can be more completely interpreted in terms of such a system than in terms of any other hitherto suggested.

VI.—Pathological Conditions.

Myasthenia gravis, paralysis agitans, lower motor neurone lesions, spastic conditions, tabes dorsalis, reflexes in "spinal" man, involuntary movements, chorea, convulsive states and contractures.

Myasthenia gravis.—In 1914 Athanasiu and Harrison [6] reported

two observations upon the voluntary electromyogram in myasthenia, i.e., that the action currents were smaller than normal and in the same direction as those of resting muscle. By contrast they reported that the direction of the action currents in normal persons during voluntary activity was the reverse of those present in resting muscle. This normal or pathological alteration in current direction has not been found by any other workers and is probably a consequence of their electrical circuits, but their diminution in the amplitude of the deflections has been since confirmed by Herzog [36] in 1917, Schaffer and Brieger [63] in 1922, and Woolf, Kleitman and Cobb [77] in 1928.

Herzog found that this was the only particular in which the electromyogram was abnormal and made the important additional observation that such patients may be fatigued to a standstill without any subjective feeling of fatigue. Woolf, Kleitman and Cobb found that the only change was one of amplitude. As fatigue advanced the frequency of the deflections remained unaltered and the amplitude, small even at the beginning, increased during the first bout of work and later slightly diminished. In contrast, when a healthy muscle was fatigued the frequency became less and the amplitude greater. Artificially induced venous stasis had no effect on the electromyogram.

Both Herzog, and Schaffer and Breiger examined the effects of electrical stimulation of the muscle at its motor point. The former found that a single stimulus resulted in many contractions, with a single action current followed by many small wavelets; the latter authors found that each stimulus gave a diphasic action current but that faradism gave a series of abnormally small deflections. Both found that the latent periods of the tendon reflexes were unaltered. They concluded that the defect lay in the muscle itself. Woolf, Kleitman and Cobb placed the essential lesion peripheral to the anterior horn cell.

The sudsidiary observations made by these authors are of great significance with regard to the site of the defects leading to the characteristic symptoms of the disease.

Herzog [36] noted the absence of a sense of fatigue even when voluntary movements had become impossible, and the absence of normal fatigue changes in the electromyogram. Woolf, Kleitman and Cobb [77] confirmed both these observations and also noted that more vigorous contractions, when these were possible, showed a greater tendency towards rhythm of their electromyogram than weaker contractions. They particularize the departure from the normal fatigue picture as showing an absence of the usual increase in amplitude and the decrease

in frequency; changes which have been shown to result from a greater degree of synchronism of the muscle units and a greater degree of volleying of the innervation impulses. It is clear, therefore, that we may summarize the findings in this disease as consisting of an absence of both the subjective feeling of fatigue and the increased synchronism of the innervation impulses to the muscle units, i.e., a failure of the normal voluntary effort syndrome which has been shown above to be associated with a central nervous mechanism rather than with the peripheral muscular or neuro-muscular apparatus.

Paralysis agitans.—Bornstein and Sanger [11], and Hoffmann Hansen and Weizsacker [40] reported that action currents are present in the resting limbs of patients suffering from Parkinsonian rigidity apart from any static tremor. These results are not confirmed by Gregor and Schilder [31], and Pritchard [52], who found that the resting limb was free from electromyographic signs of activity.

Hoffmann, Hansen and Weizsacker [40] further found that passive movements impressed upon the rigid limb without active co-operation or resistance result in periodic waves of increase of the electrical activity present in the quiescent limb. Pritchard's records of such passive movements show a series of notches upon a gradually shifting base line, these notches corresponding with the separate stages of the intermittent resistance felt in manipulation of the limb; the notches are themselves due to electrode shift at each transitory interruption of movement and each is immediately preceded by a small outburst of electrical oscillations, the curve being otherwise free from signs of action current.

Gregor and Schilder [31], 1913, showed that the static tremor corresponded to a series of tetanic contractions each of which yields two to four galvanometer deflections ("foot tremor"). In the following year Bornstein and Sanger confirmed these results. They found groups of three to five big waves at the rate of 50 to 60 per second, the groups recurring at intervals of $\frac{1}{5}$ per second with no "Nebenzacken." This rate they held to be characteristic of tremor of central nervous origin, tremor of partly myogenic origin having less frequent action currents. Rehn [58], 1922, reported that the tremor was associated with groups of large waves at the rate of 6 per second, each group containing two to three waves. Small waves at the rate of 45 per second were also present. Cobb [12], 1923, found the tremor rate averaged 5·8 beats per second, each beat was associated with a group of 2 to 4 deflections at the rate of 46 per second. Between these groups the galvanometer

was not at rest but gave smaller deflections at the rate of about 70 per second.

All these observers found voluntary movements to be associated with a series of action currents which tend to be grouped into volleys. Bornstein and Sanger [11] found these volleys to be absent at the beginning of such movements but to appear after about $1\frac{2}{5}$ second. The only difference which they found between static tremor and voluntary movement records were in respect of the amplitude, which was greater in the latter, and of the "Nebenzacken" which were absent in the former. Rehn [58] confirmed the absence of grouping of impulses at the beginning of contraction and its reappearance after contraction has progressed for a time. Pritchard [52] found in a series of patients with varying degrees of severity of affection, all stages of transition between the normal electromyogram and one in which well-marked volleys occurred at the rate of 6 to 8 per second. It was shown that such volleying is maximal when a constant tension is being actively maintained at a given degree of shortening, and disappears during stages of tension increase or progressive shortening to reappear whenever the tension is again kept constant. This disappearance of volleying with increasing tension (shortening) was not found to nearly the same extent during periods of decreasing tension (lengthening). It was shown that the tremor of movement and volleying of action currents were not, as such, signs of disturbed nervous function but occurred in normal persons in certain circumstances. They appeared in all records from patients suffering from paralysis agitans at a level of less effort and smaller tension: the difference between the electromyograms in this condition and in normal healthy persons being thus quantitative rather than qualitative.

The electromyogram in lesions of the lower motor neurone.—Richter and Ford [60] have studied the voluntary electromyogram in cases of anterior poliomyelitis, progressive muscular atrophy and syringomyelia, They came to the conclusion that such records were characterized by the following changes. The frequency of the primary waves is reduced, and may be as low as 5 to 10 per second; while the muscle is being stretched this increases up to the normal 50-per-second rate, but again decreases when the stretch is maintained. There are sometimes two rhythmic series of deflections each constant in rate and appearing or disappearing independently of the other. The amplitude, which is usually constant, is increased and there is a noteworthy lack of proportion between the large deflections and the feeble contractions. The

duration of each deflection is short, $\frac{1}{120}$ per second as compared with the normal $\frac{1}{60}$ per second. There are no secondary waves and a remarkable freedom from "Nebenzacken." It should be noted that these authors consider the latter to be due to action currents of the same origin as the primary waves and arising from elements out of phase with those which fuse to give the larger deflections. They suggest that when the reflex arc is only partially interrupted on the motor side, voluntary movement still being possible, it may be impaired to such a degree that although the tendon responses are abolished, the proprioceptive control of the innervation rhythm is maintained. Yet they hold that simple interruption of this reflex arc alone does not give the slower rhythm, for the frequency can vary in appropriate circumstances from 5 to 50 per second, the transition from one rate to another is smoother than in tabes dorsalis, and although the proprioceptive arc is interrupted in both cases the rhythm is different.

They consider that in one of their cases they were dealing with a "lesion of the motor horn cells so widespread as to leave only a few fibres intact," so that the low frequency was due to an insufficient number of impulses passing through the motor horn cells to produce an average strong contraction and thus the higher frequency of proprioceptive impulses." This they believe accounts also for the simplicity of the waves, their narrow base or short duration and the freedom from "Nebenzacken." They contrast these results with the normal electromyograms from cases of muscular dystrophy in which "what remained of the muscles could be brought into full tension" so that the usual high frequency of primary waves resulted.

The electromyogram in spastic conditions. Resting muscle.—Many investigators have reported the presence of action currents in the spastic muscles of a limb when this is fully supported and no effort at contraction is being made. This observation was made by Gregor and Schilder [31] using the calf muscles, which gave a series of small deflections in groups at a rate of 30 per second. Bornstein and Sanger [12] recorded from the biceps of the arm a similarly discontinuous stream of irregular action currents at a rate of 30 to 35 per second. In 1921 Rehn [56], using needle electrodes, found alternate periods of quiescence and activity in the records from resting muscle of a case of disseminated sclerosis, and Hoffmann, Hansen and von Weizsacker [40] obtained similar records from the contractured limbs of a case of spinal compression and from the calf muscles of a child suffering from cerebral diplegia. Rehn [56] in a similar diplegic case records a continuous

stream of tetanic "Dauerinnervation" which he considered to be characteristic of spasticity of cortical origin as contrasted with the discontinuous volleys of cases of spinal damage.

Passive movements of spastic limbs.—Gregor and Schilder [31] obtained curves from spastic muscles when passively stretched which were distinguishable from those of clonus only by the higher frequency of deflections (25, 33 per second). von Weizsacker [76] found that a perfectly normal electromyogram was given by passive stretch, and together with Hoffman and Hansen showed that the action current discharge persisted at a higher rate than in resting unstretched muscle, for long after passive movement was complete.

Voluntary movements of spastic limbs.—Samkow [62] (1912) found that voluntary movements in spastic cases were accompanied by regular action currents of longer duration ($\frac{1}{12}$, $\frac{1}{30}$ per second) and diminished frequency (11, 12 per second). Gregor and Schilder [31] found that in spastic limbs which were still under voluntary control two rhythms competed for command of the action currents. The more normal, rapid rhythm predominated in cases of slight severity, except at the beginning of movement; the slower rhythm became more prominent as the spasticity increased. The beginning of any movement was characterized by small groups of deflections at the same rate as the clonus, but during the course of the movement they were spread out and at the rate of 30 to 40 per second. Later still or with greater effort the slower rhythm returned until the complete clonus rhythm was re-established.

Bornstein and Sanger [11] confirmed these results in the following year; their groups of six, seven or eight action currents occurred only at the beginning of movement, after this the normal curve was present. From the fact that each action current lasted 0·03 second they concluded that the conduction time through the muscle was increased.

Gregor and Schilder [31] found that a tendency towards grouping of the deflections was present during voluntary movements in a case of flaccid hemiparesis; this suggests that the phenomenon is not entirely a result of increased reflex excitability and proprioceptive interference with the innervation rhythm.

Tabes dorsalis.—Gregor and Schilder [31], 1912, found that the electromyogram was altered in cases of tabes dorsalis only when the condition was advanced; the characteristics were then a diminution in frequency, an increase of the duration and considerable variation in the amplitude of the individual action currents. In addition, the intervals

between the large deflections of the galvanometer were free from secondary waves and there was found to be an inverse relation between the action current frequency and the amplitude.

In 1921 von Weizsacker [76] published a detailed examination of several patients suffering from tabes dorsalis. Certain characteristics of the electromyogram were common to all his cases, others varied from case to case. Thus he described the typical "klare Bild der vergrosten, zackenarmen, verlangsamtern Ataxierhythmus" as occurring in all severe cases, but not until a high grade of ataxy was present. In one case no action currents were detected either in resting muscles or in those passively stretched, in another the action currents present at rest were increased by passive movements. He describes a clear difference between the electromyograms of isotonic and isometric contractions. In his first case the amplitude was inconstant but increased and the rate was diminished (15 to 18 per second) with intervals free from secondary waves. Isotonic movements were associated with a greater increase in amplitude; isometric movements with a greater decrease in frequency. In his second case, a patient with far less ataxy, the isometric record was normal but that of isotonic movements showed a degree of abnormality slighter than that present in the more severe case, i.e., the rate was not so much diminished and secondary waves were present in the intervals. In a third case isometric contraction of the knee flexors gave deflections of increased amplitude at 30 per second with no secondary waves; the extensors gave the same picture but for the presence of a few secondary waves.

Richter and Ford [60], 1928, reported very different results. They found that patients with a slight degree of ataxy gave electromyograms in which the primary waves were less frequent and secondary waves were prominent. Records from those more severely affected showed no primary waves but small secondary waves of irregular frequency. They conclude that interruption of the reflex arc alone does not yield a slow rhythm, because although the arc is broken both in tabes and in affections of the anterior horn cell the electromyograms from these two cases are quite different. Their results fit in well with Fulton's generalization that "encephalization involves temporal dispersion," i.e. that relative predominance of the proprioceptive reflexes leads to greater sychronization; for here, diminution and loss of the proprioceptive impulses have led to decrease and exclusion of the primary waves, with prominence and finally isolation of the secondary waves.

v. Weizsacker [76] holds the other view, that the central nervous

discharge in tabes is of a slow, simpler (vereinfachter) rhythm unmasked by liberation from the dispersive action of the proprioceptive impulses which are normally present but lost or impaired in tabes.

It is certain that the damage sustained by the nervous system in uncomplicated but severe cases of this disease is not limited to that at the root-entry zone which leads to the loss of deep afferent impulses. There are many clinical reasons for supposing a more extensive impairment of function involving other central nervous mechanisms, and it is probably to a disturbance of motor discharge apart from the secondary effects of loss of reflex co-operation, that attention must be directed for an explanation of the electromyographic changes. If the execution of all normal voluntary movements be regarded as resulting from graded liberation from control of peripheral proprioceptive reflex mechanisms, then it is easy to see how the impairment of these mechanisms may account for such serious disturbances as are found in tabes dorsalis. But the balance of evidence is in favour of direct motor innervation in a positive sense from a higher level, and it seems probable that adjusted movements involving nice degrees of co-ordination between different muscle groups are not carried out by innervation distributing mechanisms in the cord. While it is probable that these innervation patterns have been learnt laboriously under the guiding influences of sensory proprioceptive control, it is difficult to believe that after years of practice they could be so grossly disturbed by a defect on the sensory side alone as may be seen in some cases of tabes dorsalis.

Involuntary movements and spinal reflexes.—v. Weizsacker [76] studied the electromyograms from muscles active during spinal, co-ordinated, reflex responses to cutaneous stimuli. He found that some of these were identical with those of voluntary movements. Others showed single large active currents separated by intervals of quiescence or of short tetani, the duration of individual deflections being increased. From the fact that, in spite of complete functional interruption of the spinal cord, such movements below the level of damage may yield normal "voluntary" electromyograms, he concluded that the form of the deflection curve for voluntary movements must be determined at the level of the anterior horn cells.

Persistent clonus in spastic muscle may be associated with single action currents occurring at the same rate as the contractions (Gregor and Schilder knee-clonus, 7 to 9 per second) or with small groups of action currents. (Gregor and Schilder hand- or ankle-clonus 2 to 4 waves, Bornstein and Sanger [11] ankle-clonus 3 to 5 waves at $\frac{1}{5}\frac{1}{0}$ second intervals). Gregor and Schilder thought that there was probably only

one motor discharge per beat, and that interference at the muscle equator of action currents arising in different parts of the muscle lead to its apparent reduplication in some cases. Hoffmann, Hansen and Weizsacker [40] found that each clonic beat from the calf muscles in a case of cerebral diplegia was associated with a volley of several large deflections. They also made the observation that each tendon-jerk obtained from a spastic muscle is followed by a temporary inhibition of the pre-existing spastic discharge.

Involuntary movements—chorea.—Bornstein and Sanger [11] described for chorea an electromyographic picture having "an extraordinary resemblance" to that of paralysis agitans, in that the action currents of increased duration (0·03 second) occurred at a lower frequency (30 per second). From these observations they concluded that the conduction time in the muscle, from the region of nerve-entry to the extremities, was increased. However, records differing in no wise from those of normal voluntary movements have been obtained and described by Gregor and Schilder [31], Salamonson [61] and Cobb [12].

Rehn [57] describes records from a case of torsion dystrophy as being normal in appearance except for a tendency for the occurrence of prolonged groups (lasting $\frac{3}{4}$ to $1\frac{3}{4}$ seconds) of regular action currents at 45 per second.

Although Fröhlich and Meyer had found no action currents associated with maintained cataleptic attitudes when using skin-pad electrodes, Rehn [56] obtained normal electromyograms from the same condition with needle electrodes, a result confirmed by Hoffmann and Weizsacker. Normal records from cases of hysterical movements have been obtained by Rehn [56] and Cobb [12].

Convulsive state and contractures.—That the muscular cramps or spasms associated with some cases of amyotrophic lateral sclerosis give electromyographic records similar to those of normal voluntary movements has been reported by Hoffmann [39], and Hoffmann, Hansen and Weizsacker [40], although Bornstein and Sanger [11] had previously found action currents absent in this condition.

Tetanic spasm has been described as unassociated with any form of action current by Salamonson [61] and Semereau and Weiler [64].

VII.—Summary.

(1) A short account has been given of the previously recorded electromyograms of normal voluntary movements in man. These have been compared in respect of the amplitude, form and frequency of their individual deflections.

(2) An attempt has been made to outline the principles governing the interpretation of such records in terms of muscle activity and expressed in terms of the electrical and mechanical components of such activity.

(3) The principles underlying the known distribution of the electric field about active, simple muscle strips surrounded by a conducting medium have been applied to the case of skeletal muscle voluntarily made active in the intact limb.

(4) The inferences permissible with regard to the motor discharge from the central nervous system during voluntary movements have been stated and the factors governing this discharge examined.

(5) A suggested explanation for the so frequently observed dominant 50-per-second rhythm has been advanced.

(6) The influence of fatigue upon the action currents of voluntary movements has been detailed. The effects of such fatigue have been correlated under the term "effort syndrome" and the other conditions and modes of occurrence of this effort syndrome stated.

(7) An attempt has been made to determine the site of the mechanism responsible for the phenomena of the effort syndrome. As a result of the inquiry the existence of a mechanism having certain properties and located in a certain part of the reflex system has been postulated.

(8) Short summaries have been made of the recorded characteristics of the voluntary electromyograms obtained from patients suffering from different pathological conditions.

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PROGRESSIVE HYPERTROPHIC POLYNEURITIS, WITH CASE REPORTS.

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PROGRESSIVE hypertrophic polyneuritis was first described in 1899 by Goubault and Mallet [6], and four years later was designated as a new clinical entity by Dejerine and Sottas [4], with whose names the condition is usually associated.

Pathological confirmation of the diagnosis in a number of published cases shows that the clinical picture varies within very wide limits, while the pathological changes themselves vary both in detail and in degree. Thus there may be no family history of similar disease, as in cases reported by Boveri [2] and Dide and Courjon [5]; the disease may begin in adult life (De Bruyn and Stern [3], Harris and Newcomb [7], and Long [8]); the course may be characterized by remissions [7]; and the degree of peripheral nerve enlargement may be slight [3, 4, 7], or very great, as in the cases reported by Boveri and Souques and Bertrand [11].

Pupillary abnormalities, nystagmus and kypho-scoliosis, originally described as features of the disease, have been present in a few cases only; exophthalmos in one family [2] and persistent diarrhœa in cases reported by Pierre Marie [9] introduce additional features.

The wide variations in the symptomatology make any attempt at describing types of the disease valueless, and it must be acknowledged that there are only two constant clinical features, namely, muscular atrophy and the peculiar enlargement of the peripheral nerves.

The cases here described illustrate again the variability in the clinical picture. Case 1 shows extreme wasting and enlargement of all the peripheral nerves in a young adult; in Case 2, on the other hand, wasting of one hand was noticed for the first time at the age of 38; pes cavus had been present since childhood, and five relatives show neurological changes probably related to the same condition.

Cases 1 and 2 have been fully examined as in-patients at the National Hospital, and the diagnosis has been confirmed by biopsy. The other five patients have been visited at home, and are not under medical supervision.

Case 1.—G. F., female, aged 32, was admitted to hospital under Dr. Macdonald Critchley on February 7, 1930, complaining of progressive weakness and numbness of the limbs with wasting of the hands and feet.

There was no history of illness in the parents, who had one other child; this child had tuberculous cervical adenitis, but is now a healthy and active man. No history of illness in more distant relations is available.

The patient suffered from measles, whooping-cough and chickenpox as a child, and had a severe attack of influenza in 1918 (when the temperature reached 105° F.). She had been troubled with cough all her life; there had never been sputum or hæmoptysis, but there was an attack of pain in the chest several years ago. For the past few years there had been dyspnœa on exertion and steady loss of weight, though she had never weighed more than 8 st. Menstruation was regular until she was 24, when there was amenorrhœa for four years; the menses were regular from 1926 to 1929, but since then there has been complete suppression.

The first definite symptom of the present illness was weakness of the legs, first noticed five years ago. About a year later weakness of the hands developed. This weakness steadily progressed, and was always more marked in the summer or if she was in a hot room. For several years there had been attacks of stabbing pain all over the body, but most marked in the hands and feet, and worse in the summer months. Numbness of the hands and feet had been progressive throughout, and she stated that she knew nothing of her feet unless she could see them; she was unable to stand on her bare feet. The colour of the feet, and to less extent of the hands, varied from purple to a vivid red, according to the room temperature. She had noticed a flickering sensation under the skin and had seen the flickering from time to time. There had been no sphincter disturbance.

Examination.—The patient is a small, emaciated woman (weight 5 st. 8 lb.). She is intelligent and shows no abnormality of higher cerebral functions. The fundi and pupillary reactions are normal, there is a rapid, ill-sustained nystagmus on looking to the right, and a mild degree of bilateral ptosis. The lower part of the right side of the face is weak both in voluntary and emotional movements. The other cranial nerves show no abnormality. There is generalized wasting of both arms in proportion to the general emaciation, with well-marked distal accentuation which becomes extreme in the intrinsic hand muscles, especially on the thenar side. Grip is moderately good on both sides, but there is extreme weakness of all the intrinsic hand muscles. There is a fine rapid tremor of the outstretched hands as well as a slower, coarser and more irregular movement of the fingers. Both arms are slightly hypotonic; the finger-nose test is accurately carried out.

There is extreme wasting of the legs, which begins at the junction of the middle and lower thirds of the thigh and increases towards the periphery. The feet are inverted and plantar flexed. There is a full range of active and passive movements at the hips and knees, though all movements are weak. There is practically no active movement at the ankle-joints, and passive dorsiflexion is limited and painful. Tone is diminished in both legs. The heel-knee test is carried out on both sides with slight inaccuracy. The trunk muscles are strong and are not wasted. Fibrillation is occasionally seen in the arms and thighs. Gait is steady and progress fairly rapid, but the feet are lifted very high; there is a good deal of unsteadiness when turning and well-marked Rombergism.

There is considerable sensory disturbance. Pain sensibility is practically lost over the arms and fore-arms and between the knees and ankles, though there is little impairment over the hands, feet and trunk. Light touch is everywhere normally appreciated. Temperature sensibility is only lost over the feet. Two-point discrimination is very defective over the hands and feet, and vibration is not appreciated below the knees. The sense of position and of passive movement is very defective in the toes, but is not impaired in the fingers. Deep pressure pain is lost below the knees. There is intense vaso-motor change in the feet, with a less marked change and excessive sweating in the hands.

There is a great and generalized enlargement of the peripheral nerves; the nerves are hard and painless and are especially noticeable in the upper arm and neck. The internal cutaneous nerve of the arm is visible as a thick, beaded cord (fig. 1) and there is extreme enlargement of the ulnar and external popliteal nerves.

All the tendon-jerks are very sluggish and the supinator and ankle-jerks are absent. The abdominal and plantar reflexes could not be obtained.

The chest is long and narrow with impaired movement on the right side; the percussion note is impaired at both apices, and there is prolonged expiration with numerous crepitations all over the chest.

The nails are lilac tinted.

X-rays show opacity at the right apex with numerous areas of infiltration throughout both lungs.

Special investigation.—Blood-pressure is 130/84 mm. The red-cell sedimentation 7·5 in one hour. The blood-Wassermann reaction is negative. The resting blood-sugar is 100 mgm. per cent. with normal curve after the ingestion of glucose. There was no sputum and no tubercle bacilli in the fæces. "All the muscles of the arms, fore-arms and hands show partial reaction of degeneration; the faradic response can be obtained with large currents; the galvanic response is typically sluggish. The muscles of the thigh also show partial R.D. The muscles of the legs show complete R.D., no faradic response being obtained with bearable currents" (Dr. L. W. Bailey).

On admission to hospital the evening temperature was 99° F. and the pulse-rate 100. Following the administration of potassium iodide, the temperature

rose to 101° F. and the pulse-rate to 120; cough increased but remained unproductive. On withdrawing the iodide the temperature and pulse-rate fell to their previous levels.

Appetite improved on giving insulin (5 units t.d.s.), but weight remained stationary.

A biopsy was performed under local anæsthesia and a piece resected from the internal cutaneous nerve of the left arm. This was irregularly thickened, varying from 5 mm. to 3 mm. in diameter (fig. 2). On cross-section about three strands of homogeneous gelatinous tissue were seen; the finely fibrillar structure of the normal nerve was completely lost.

Celloidin sections stained with van Gieson showed each of the three strands to be surrounded by a definite, and probably normal, perineural sheath; and in this lay sparse nerve-fibres considerably separated from one another, each being surrounded by a thick zone of concentrically arranged connective tissue fibres derived from the endoneurium. Few nuclei were seen in these zones, but there were usually one or more nuclei near the centre in which was a clear ring representing the myelin sheath and axis-cylinder. A few nuclei and scattered strands of collagen running longitudinally were seen in the tissue separating the nerve-fibres, the rest of which appeared structureless and took on a faint yellow colour with this stain. When stained by the Weigert-Pal method, most of the nerve-fibres showed a definite, though thin, myelin sheath, and in longitudinal section this was seen to run uninterrupted when present (fig. 3). Occasionally two myelinated fibres were seen to run in one endoneural sheath. In the interstitial spaces were seen large irregular cells with clear cytoplasm and large irregular nuclei; these appeared to be of endothelial origin.

Case 2.—W. I., male, aged 38, was admitted to hospital on March 24, 1930, under Dr. Kinnier Wilson, complaining of wasting of the right hand.

He was quite healthy as a small boy and was able to play football and other games. After he left school (aged 14) his toes began "to contract" and his feet to change shape. He then found that he could not run so well as formerly and that he was less steady on his feet. This disability in his feet has not progressed during the last twenty years.

He drinks about one glass of beer per week. He is a storekeeper and frequently handles lead while at work.

Three and a half months ago, coincident with the onset of colder weather, he noticed some numbness of the index finger of the right hand, and that when his hands were cold his pen used to slip out of position. There was also some wasting of the hand about which he consulted his doctor. No symptoms suggestive of plumbism were present. He remains quite fit for his work and spends all day on his feet. There have been no other symptoms.

Examination.—The patient is a healthy looking man of spare physique and average intelligence. There is no abnormality of speech or of the higher cerebral functions. The fundi and pupillary reactions are normal. The

external ocular movements are full and there is no ptosis or strabismus. Fine horizontal nystagmus is present on looking both to the right and to the left, the quick component being in the direction of gaze. (This is not constantly present.) The other cranial nerves show no abnormality.

There is considerable wasting of all the small muscles of the hands and of the fore-arms, especially in their distal halves. This is much more pronounced on the right side. Power is diminished in proportion to the degree of wasting. There is no weakness or wasting of the upper arms.

The outstretched arms maintain their posture, but small movements of the fingers are seen on both sides and are associated with fibrillary twitching in the muscles. Co-ordination tests are accurately performed. No abnormality of muscle tone was detected. There is marked upper dorsal scoliosis, convex to the right. The trunk and abdominal muscles show no abnormality.

There is bilateral pes cavus. Wasting of the anterior tibial and peroneal muscles is marked in both legs, and this is associated with marked weakness. There is no weakness of movement at the hip or knee on either side. Co-ordination is good. No fibrillation is seen in the lower extremities.

"All the intrinsic muscles of both hands show partial R.D. as well as the flexors of the right fore-arm. The extensors of the right fore-arm gave normal responses. In the right leg the tibialis anticus, peronei and extensor digitorum brevis show complete, and other muscles partial, R.D. All muscles of the left leg show partial R.D." (Dr. L. W. Bailey.)

All the tendon-jerks are absent. The abdominal reflexes are present, but sluggish. Both plantar reflexes show frank extensor responses (no weakness of the toe flexors).

There is no sensory loss to pin-prick or cotton-wool. Temperature sensibility, sense of position and passive movement are all normal and vibration is normally appreciated. There is no tenderness of the muscles, but the soles of the feet are unusually sensitive.

There is considerable enlargement of all the peripheral nerves. The median nerves in the cubital fossæ are particularly large, and the course of some of the cutaneous nerves of the fore-arm is visible through the skin. The nerves are firm and their surface feels slightly irregular. They are not tender on firm pressure.

The gait is steady and brisk. There is a drop-foot on the right side. Romberg's sign is not present.

Examination of the cardiovascular, respiratory and genito-urinary systems reveals no abnormality.

Special investigation,—The cerebrospinal fluid is clear and colourless, cells 4 per c.mm. (20 per cent. large mononuclears), total protein 0·10 per cent., Nonne-Apelt test weakly positive, Pandy test negative, Lange 0011100000, Wassermann reaction negative. Blood Wassermann reaction negative. X-rays show absence of cervical ribs; a fractional test meal shows complete achlorhydria (total chlorides 0·3 per cent. after one hour). The blood-picture is normal.

PLATE XVII.

FIG. 1 (*Case* 1). —Showing the nodular enlargement of the internal cutaneous nerve.

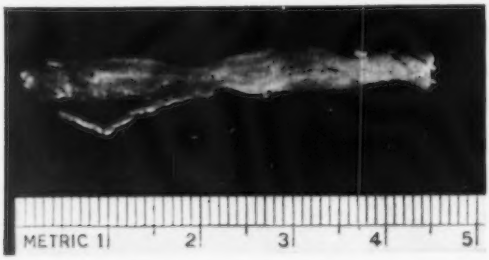

FIG. 2 (*Case* 1).—Resected portion of internal cutaneous nerve.

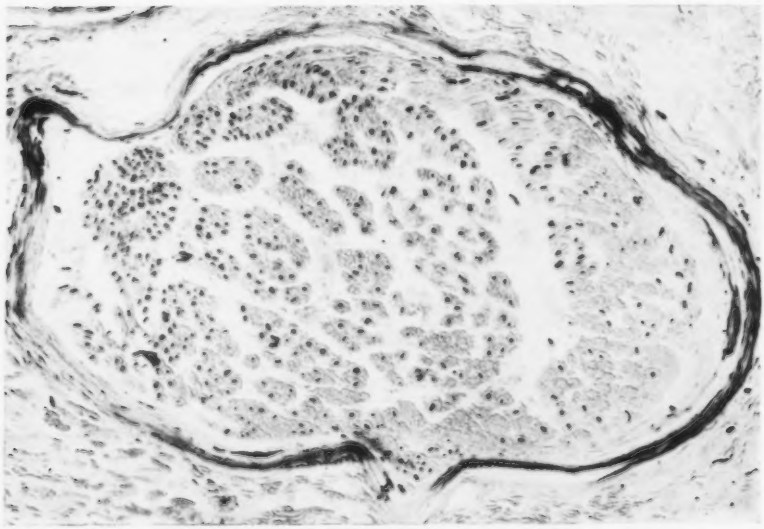

FIG. 3 (*Case* 1).—Cross section of nerve. Weigert-Pal stain.

To illustrate paper by W. Ritchie Russell and Hugh G. Garland.

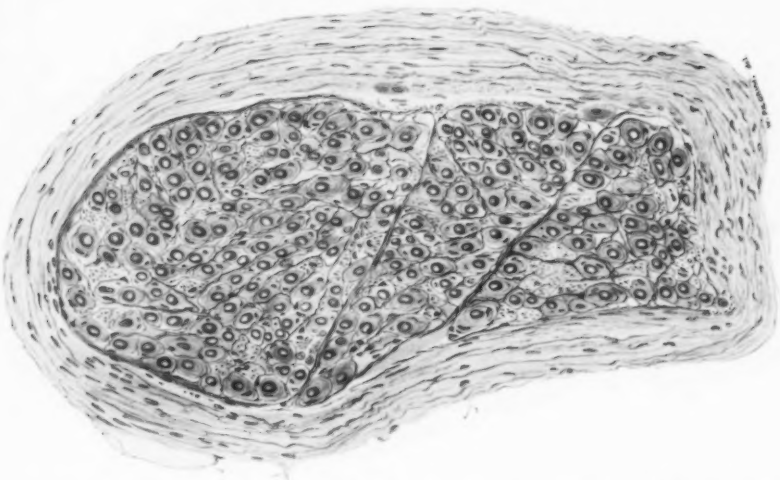

FIG. 4 (*Case* 2).—Cross-section of a bundle in the nerve resected. To show the thickening of the endo- and peri-neurium. Scharlach R. × 200.

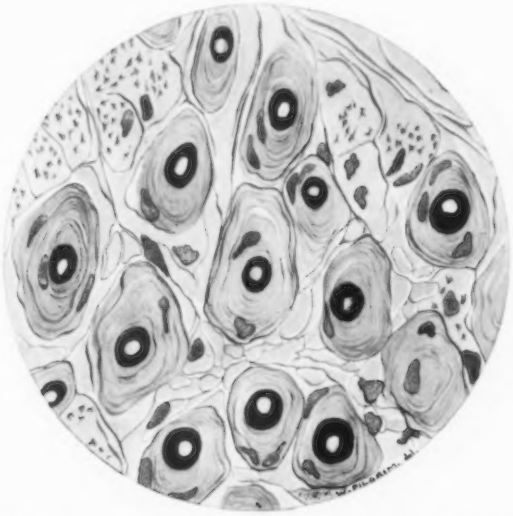

FIG. 5 (*Case* 2).—A portion of the same bundle under a higher magnification to show the nuclei and concentric lamellæ in the endo-neurium. Scharlach R. × 1,000.

To illustrate paper by W. Ritchie Russell and Hugh G. Garland

A portion of the internal cutaneous nerve of the left arm was removed under local anæsthesia.

Frozen sections show some separation of the individual nerve-fibres by fluid or mucoid infiltration. Each individual myelinated fibre is also surrounded by several concentric layers of connective tissue capsule which form a zone at least as wide as the transverse diameter of the myelin sheath. In celloidin sections, in which the nerve-fibres are more shrunken together than in frozen sections, the appearance is that of isolated myelinated fibres separated from one another by dense and fairly wide envelopes of fibrous tissue (endoneurium). The perineurium is much thicker than normal. The interstitial spaces appear to contain more nuclei than are seen in Case 1, in which the condition is more advanced (figs. 4 and 5).

Family history.—A history of some abnormality apparently related to this patient's condition was found in eight other members of his family. Five of these have been examined (fig. 6).

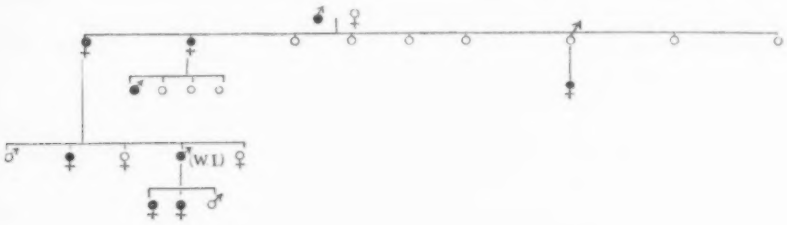

FIG. 6.

The patient's mother's father died of a stroke at the age of 65. He had curvature of the spine and used to lift his feet high when walking. His feet were very small, but no deformity was noticed. Patient's mother had nine brothers and sisters, none of whom was in any way abnormal.

Case 3.—The mother of Case 2 is now aged 65; she has had difficulty in walking ever since she had a severe attack of rheumatic fever when aged 15. She has had to wear special boots since that time owing to deformity of the feet. She has suffered severely with "arthritis" for twenty years, which has necessitated her using a bath chair. She has had stabbing pains in the hands for twenty years.

Examination.—The pupils react sluggishly to light. The outstretched hands are tremulous. All the tendon-jerks are absent, and the plantar reflexes are indefinite. There is very pronounced pes equino-varus on both sides, and gross arthropathy with disorganization of both knee-joints, allowing about 40°

of lateral movement, and extreme genu valgum on attempting to stand. Passive movement of these joints causes little pain and they resemble very closely the Charcot's joints of tabes dorsalis. There is considerable arthritis of the joints of the fingers, causing awkwardness and weakness of fine movements of the fingers. No fibrillation was seen. No sensory loss to pin-prick, nor to light touch, nor loss of appreciation of passive movements was detected.

This patient has five children alive:—

Al., male, who is quite normal.
Al., female, aged 42, affected (Case 4).
E., female, aged 40, quite normal (with children).
W., male, aged 38, affected (Case 2).
Et., female, aged 34, quite normal (with children).

Case 4.—Al., female, aged 42, has had difficulty in walking since the age of 12, together with deformity of the feet. There has been wasting of the right hand for the past seven years. Four years ago she had severe pains in both hands and fore-arms for seven weeks, and following this great weakness of the hands developed. This has improved considerably since then. Five years ago there was some numbness in her right hand.

Examination.—The pupils are of average size, but react sluggishly to light. There is ill-sustained nystagmus on lateral deviation of the eyes. The cranial nerves show no other abnormality. There is pronounced wasting of all the small muscles of the hands, especially of the first dorsal interossei; the right hand is more affected than the left. There is also wasting of the distal half of each fore-arm.

There is a marked degree of bilateral pes cavus and there is some wasting of the muscles below the knees. The feet are very cold. All the tendon-jerks are absent. The plantar responses are both flexor.

There is some diminution to pain sensibility over the right foot, and the lower part of the leg, but there is no other sensory loss. The accessible nerves are easily palpable and are probably larger than normal; they are hard and not at all tender on deep pressure.

Case 2 has three children:—

J. I., female, aged 13 (Case 5).
C. I., female, aged 11 (Case 6).
A. I., male, aged 7, who was examined and appeared to be quite normal.

Case 5.—J. I., female, aged 13, is quite healthy, but her hands have always been shaky, especially in the morning. She is apt to fall while playing games. An operation for bilateral pes cavus was performed at the age of 11½ years.

Examination.—She is well-developed physically and mentally. There is some ill-sustained nystagmus on looking to each side. The outstretched hands are tremulous and are a little unsteady in co-ordination tests.

All the tendon-jerks are absent; the plantar responses are flexor. No sensory loss was discovered.

The accessible nerves are easily palpable: they are hard and are not tender on deep pressure; they are probably not enlarged.

Case 6.—C. I., female, aged 11, has always enjoyed good health. She had "St. Vitus' dance" at the age of 2, but has had no other illnesses. The mother thinks she is normal in every way.

Examination.—She is a well-developed child, both physically and mentally. The pupils are normal. There is ill-sustained nystagmus on looking both to the right and left. No tremor or other motor disturbance was observed.

All the tendon-jerks are absent; the plantar responses are both flexor. There is no deformity of the feet and no sensory loss could be detected.

Case 7.—B. W., female, is a first cousin of W. I. (Case 2), being the daughter of his mother's brother. She has no brothers or sisters; her mother has had no miscarriages. The patient became unsteady on her feet at the age of 7, and has become steadily worse. She has developed curvature of the spine. An operation was performed on both feet when she was 14, but there was no improvement. A year ago she had an acute febrile illness with swelling of both knees, and on recovering from this she noticed numbness and "pins and needles" in both hands, and experienced some difficulty in using them. This disability has improved since then. She has no pain now except in her feet.

Examination.—The patient is thin and pale; she is normally developed mentally. The pupils are normal. There is ill-sustained nystagmus on looking to the right and to the left. The other cranial nerves show no abnormality. There is no obvious wasting in the upper limbs, but there is some tremor of the outstretched hands, especially the right. Power in all muscle-groups is fair. She can hyper-extend her fingers at the metacarpo-phalangeal joints to a right angle. She has marked kypho-scoliosis and an extreme degree of club-foot on both sides. The range of movement at the ankle-joints is very small, the dorsiflexors of the feet being especially weak. There is no gross wasting of the legs.

All the tendon-jerks are absent; the plantar reflexes are doubtful. No sensory loss to pin-prick was discovered; sense of position is normal. Light tactile stimuli are not normally appreciated over irregular areas of her legs.

All the palpable nerves are enlarged; they are hard, and are not tender on deep pressure.

This patient's mother's sister has four children; none of these was examined, but one, aged 36, "has something wrong with his feet."

Discussion.

Various opinions have been expressed with regard to the nature of this disease and its relationship to other familial conditions.

Marinesco, Raymond, Bedaschi and Long [8] have regarded it as a variety of Charcot-Marie-Tooth's disease. On the other hand, Souques

[10] and de Bruyn and Stern [3] have claimed a separate identity for hypertrophic neuritis, both clinical and pathological.

The close resemblance to peroneal atrophy is shown by Case 1, but there was also the rosary-like enlargement of the nerves which Bielschowsky [1] considered to be one of the features which related these cases to von Recklinghausen's neuro-fibromatosis. Enlargement of the peripheral nerves is a well-known phenomenon in von Recklinghausen's disease and has been described by Thomson [12] in his monograph on this subject.

The presence of a definite extensor plantar response in Case 2, and of a doubtful response in Cases 3 and 7, suggests a possible relationship between hypertrophic neuritis and the familial ataxias.

Despite the close relationship to other diseases which is sometimes apparent, it seems advisable to regard the condition as a separate entity until more conclusive evidence of its relationship to other diseases is established.

SUMMARY.

(1) Progressive hypertrophic polyneuritis appears to be a clinical and pathological entity.

(2) The symptomatology is extremely variable. Enlargement of the nerves and wasting of muscles are the only constant changes.

(3) Seven cases are described, in two of which the diagnosis has been confirmed by microscopic examination of the nerves.

(4) In this series nystagmus and absence of tendon-jerks are common to all the cases.

We wish to thank Dr. Kinnier Wilson and Dr. Macdonald Critchley for permission to publish these cases, and to express our indebtedness to Dr. J. G. Greenfield, who has directed the pathological investigations and kindly supplied the photomicrographs.

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Notices of Recent Publications.

Les Plexus Choroïdes. Par Dr. Nathalie Zand. Pp. 140. Paris: Masson et Cie. 1930. Price 44 francs.

This essay on the choroid plexus is divided into three chapters which deal respectively with the anatomy, physiology and pathology of the plexus and the structures with which it is in intimate relationship, especially the cerebrospinal fluid and the ependyma.

The chapter on anatomy is concise and straightforward enough, but in attempting to deal with the physiology of the choroid plexus and cerebrospinal fluid, the author has become involved in a maze of conflicting and even contradictory theories and observations from which she makes but a half-hearted attempt to extricate herself. Much of this confusion might have been avoided if she had paid as much attention to the latest work as she has to some older observations. The chief criticism which will be levelled against this part of the book is that it completely neglects the Donnan membrane theory in its relation to cerebrospinal fluid circulation, on which so much recent work is based. In the chapter on pathology some useful facts are collected, but the limited nature of the author's material has prevented her from adding much of importance. She is satisfied, however, that there is little qualitative difference in the effect which different diseases and intoxications produce on the choroid plexus. The chief differences lie in the fact that acute conditions affect the epithelium and more chronic ones the vasculo-conjunctival structure of the plexus. This chapter contains some material which is unfamiliar to most neurologists, and is of more value than the rest of the book.

Teknik der Microskopischen Untersuchung des Nervensystems. von W. Spielmeyer. 4th Auflage. Pp. 168. Berlin: Julius Springer. 1930. R.M. 11.40.

The fourth edition of this valuable laboratory handbook preserves the format of the last edition, and is little increased in size, in spite of the addition of much new material. This has been made possible by the sacrifice of older methods as they are replaced by newer ones. For example, old-established methods for staining neuroglia, such as those of Bevan Lewis and of Bethe, have been omitted to make room for the various modifications of Hortega's silver methods. We are glad to see the inclusion of several English methods which were absent from the last edition, such as Anderson's Victoria blue

stain for neuroglia, Perdrau's silver stain for fibroglia and Laidlaw's method for peripheral nerve-fibres. Another welcome addition is the enlargement of the section on the staining of spirochætes in nervous tissue.

In a book of this size it is impossible to include everything, and two notable omissions are Busch's osmic acid method for myelin degeneration, and the use of formol saline or of Kaiserling's fluid as fixatives. It is indeed surprising to see that a solution of formalin in water is still recommended as a fixative for the nervous system. But every laboratory worker has his individual preferences, and it appears to be impossible, especially with the nervous system, to standardize technique. It is therefore all the more valuable to have a description, in a compact and practical form such as this, of the technique used in the Kaiser Wilhelm Institut at Munich.

Nouveau Traité de Psychologie. Par GEORGES DUMAS. Tome I. Notions préliminaires. Introduction. Méthodologie. Pp. 425. Paris: Alcan. 1930. Price 75 francs.

This is the first volume of a series in which normal psychology (in eight volumes) and pathological psychology (in two) are to be presented by such noted French authorities as Janet, Claparède, de Clerambault, Séglas, Piaget, Pieron, Levy-Bruhl and many more.

The present volume is mainly introductory. Articles on the place of man in the animal series and on the data of anthropology are tokens of thoroughness in the general plan of the work—the first by Perrier, the second by Rivet. The physiology of age and sex is dealt with by Champy, Pézard's work in this field naturally receiving much attention; conformably to the present state of knowledge the writer confines himself largely to comparative observations, with none of those hardy incursions into conjecture and ill-founded affirmation which are met with, especially among psychiatrists, in this matter. Lapicque, known for his distinguished research into chronaxy, deals with the general physiology of the nervous system, chiefly in the light of his special studies. Tournay's competent article on the special physiology of the nervous system is followed by a chapter of great interest by Henri Wallon on the biological problems of consciousness; the influence of von Monakow is here strongly in evidence, and the "phenomenology" of Jaspers is discussed in some detail. At this point the "notions préliminaires" come to an end, and there follow the first two chapters of Book I—an introduction to psychology by G. Dumas, the general editor of the series, and André Lalande's conspectus of methods and aims in psychological investigation.

In this volume the discussion ranges, as one would expect, from philosophy to physiology, but it is balanced and restrained. The bibliographical lists are by no means restricted to French works. The volume is well illustrated but has no index.

The Psychology of Insanity. By BERNARD HART. Pp. 176. Cambridge University Press. 1930. Price 3s.

Dr. Hart's well-known little book appears in the fourth edition, little changed from its first form of eighteen years ago. An introduction has been added dealing chiefly with the development of psychopathology as expressed in the teaching of Janet and Freud, and most of the footnotes of former editions have been taken up into the text. It is scarcely necessary to say that the book fulfils its modest aims admirably; intended for the beginner and the layman, its simplicity is a merit, even though it fails to indicate the great complexity of the problems concerned.

The Basis of Epilepsy. By EDWARD A. TRACY, M.D.(Harvard), Director of the Epileptic Clinic at Forsyth Dental Infirmary, Boston. 1930. Rich. G. Badger, Boston, U.S.A. Pp. 88.

The thesis of this work is based upon the observation of the intense colour and duration of the white streak of vaso-constriction that followed stroking of the skin in epileptics with grand and petit mal. The time variability in addition to the above were noticeably different from that in normal control cases.

The book contains the time observations in detail of the whole investigation, and is illustrated with pictures of the reaction, and of the methods for eliciting it. The author argues from these findings to the conclusion that epilepsy must be regarded as a clinical entity having vaso-motor capillary constriction in the brain as the physio-pathology of the disorder.

Medizinische Psychologie. von ERNST KRETSCHMER, Professor für Psychiatrie in Marburg. 1930. Leipzig: Geo. Thieme Verlag. Pp. 259. Price 16 marks.

This is the fourth and enlarged edition of this now celebrated volume. The book is written in the light of fundamental principles. The well-known reaction type theory of its author is fully discussed, and his biological theory of hysteria is also given in extended form. This book only awaits translation to make it more widely and deservedly accessible to all English psychiatrists.

The Mental Development of the Child, By KARL BUHLER, Professor in the University of Vienna. 1930. London: Kegan Paul. Pp. 170. Price 8s. 6d.

The author has brought together in a brief space the contents of a larger work on the psychology of the child. It attempts to trace the biological foundations of child psychology, and in consequence passes systematically through child development, pausing to consider inheritance of mental qualities,

and dealing at length with the perceptual world of the child. Imagination is considered in general, and in particular by reference to its operation in fairy tales. A particularly good chapter is devoted to the development of drawing. The shortcomings of the book are obvious in the treatment of the affective life and the inner life of fancy. The contact with other human beings, particularly the parents, who have the first and lasting influence on the instinct and emotional life both in increasing and in deflecting affectivity, is not dealt with at all. It is now generally admitted that some of the striking features of child life spring from these affective relations to the parents. The author apparently has no use whatever for this approach, and entirely ignores the work of Freud.

Eidetic Imagery, and Typological Methods of Investigation. By E. R. JAENSCH, Professor of Psychology in the University of Marburg. 1930. London: Kegan Paul, Library of Psychology. Pp. 136. Price 7s. 6d.

The classification of human beings according to their imagery exercised the mind of Francis Galton. Little did he appreciate, however, the extent to which the study of imagery under laboratory conditions could be applied to the wider fields of medicine and psychology. In this work Professor Jaensch, who has with his brother been a pioneer of experimental work on a type of imagery, the eidetic, has outlined the method of detecting this type of imagery. The eidetic image differs from both after images and memory images in that it is more closely related in form and behaviour to actual perception. This typological study is no mere exercise in academic psychology, for it digs deeply into the psycho-physics of the human organism. For example, it is found that there is a variability of the eidetic image in relation to calcium metabolism. So much is this the case that the author has discovered a relationship between this type of imagery and tetanoid and basedowian syndromes. The image variations thus studied are not to be regarded as pathological, but are due to functional peculiarities of the autonomic system characteristic of certain youthful stages of development.

Linked with the Jungian classification of mental types, and with Kretschmer's type classification, the application of Jaensch's work to both pedagogics and psycho-pathology promises much fruitful discovery.

The Laws of Feeling. By F. PAULHAN. 1930. London: Kegan Paul. Pp. 208. Price 10s. 6d.

This carefully reasoned essay was not sufficiently known in England, and there is now no excuse for English readers to ignore a theory of feeling which must have a profound effect on the theories of emotion. Hitherto English psychology has been held by the theory of MacDougall. The thesis is, broadly,

that feelings as well as emotions are not the accompaniments and tone colourings of the instincts in action, but are due to arrested tendency. It rests still more profoundly on the author's view that man is essentially an unadapted animal, and that feeling and emotion are the results of this maladaptation.

Psycho-pathology, from much of its data relative to repression and the symptom complexes that spring from it, lends support to this theory: and neurology, too, particularly the relationship of autonomic function to the thalamus, gives facts corroborative of this theory.

This is a book that no thoughtful-minded psychiatrist can afford to ignore.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I to XXIII inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

To those who are not members of the Neurological Section of the Royal Society of Medicine the price is 8s. 6d. net, and the volume may be obtained through any bookseller.

EDITOR.